# A STUDY ON PREVALENCE OF METABOLIC SYNDROME IN PATIENTS WITH ACUTE CORONARY SYNDROME

*Dissertation submitted to*

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*In partial fulfillment of the regulations*

*for the award of the degree of*

**M.D. BRANCH - I**

**GENERAL MEDICINE**

**GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL**

**THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITYCHENNAI, INDIA.**

**APRIL 2011**

# CERTIFICATE

This is to certify that the dissertation titled **"PREVALENCE OF METABOLIC SYNDROME IN PATIENTS WITH ACUTE CORONARY SYNDROME"** is the bonafide original work of **Dr.A.MEENAKSHI,** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in April 2011. The Period of study was from May 2008 to May 2010.

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## DECLARATION

I, **Dr.A.MEENAKSHI,** solemnly declare that dissertation titled **"PREVALENCE OF METABOLIC SYNDROME IN PATIENTS WITH ACUTE CORONARY SYNDROME"** is a bonafide work done by me at Government Stanley Medical College and Hospital during May 2008 to May 2010 under the guidance and supervision of my unit chief **Prof.DR.K.H.NOORUL AMEEN** Professor of MEDICINE Government Stanley Medical College and Hospital, CHENNAI.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine april 2011.**

Place : Chennai **A.MEENAKSHI**

Date :

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## INTRODUCTION

Metabolic syndrome is one of the health issues of this century. It is a constellation of physical conditions and metabolic abnormalities commonly occurring together, that increases the individuals risk for the development of type 2 diabetes mellitus and cardiovascular disease. If the current trend continues the premature deaths and disabilities resulting from these conditions will increase the financial burden in developed and developing countries.[1]

As the prevalence of metabolic syndrome is high worldwide and increasing day by day due to sedentary lifestyle the findings of the present study has important implications for clinical practice emphasis must be placed on the intake of balanced diet and the control of lipid level particularly that of triglycerides.

## AIM

To ascertain the prevalence of metabolic syndrome in patients with acute coronary syndrome and to find out the association of each component of metabolic syndrome with acute coronary syndrome.

# REVIEW OF LITERATURE

## Definition of metabolic syndrome

It is defined as clustering of cardiovascular risk factors in an individual which predisposes the person to a greater risk of developing type 2 diabetes mellitus and cardiovascular disease.

It was first termed as syndrome X in 1988.

Main feature of metabolic syndrome includes insulin resistance.

Criteria for diagnosis

International disease federation

WHO criteria

National cholesterol education panel ATP III guidelines.

# HISTORY OF METABOLIC SYNDROME

The term "metabolic syndrome" dates back to at least the late 1950s, but came into common usage in the late 1970s to describe various associations of risk factors with diabetes that had been noted as early as the 1920s

The Marseilles physician Dr. Jean Vague, in 1947, observed that upper body obesity appeared to predispose to diabetes, atherosclerosis, gout and calculi.

Avogadro, Crepaldi and co-workers described six moderately obese patients with diabetes, hypercholesterolemia, and marked hypertriglyceridemia all of which improved when the patients were put on a hypocaloric, low-carbohydrate diet.

In 1977, Haller used the term "metabolic syndrome" for associations of obesity, diabetes mellitus, hyperlipoproteinemia, hyperuricemia, and hepatic steatosis when describing the additive effects of risk factors on atherosclerosis.

The same year, Singer used the term for associations of obesity, gout, diabetes mellitus, and hypertension with hyperlipoprotenemia.

In 1977 and 1978, Gerald B. Phillips developed the concept that risk factors for myocardial infarction concur to form a "constellation of abnormalities" (i.e., glucose intolerance, hyperinsulinemia, hypercholesterolemia, hypertriglyceridemia, and hypertension) that is associated not only with heart disease but also with aging, obesity and other clinical states. He suggested there must be an underlying linking factor, the identification of which could lead to the prevention of cardiovascular disease; he hypothesized that this factor was sex hormones.

In 1988, in his Banting lecture, Gerald M. Reaven proposed insulin resistance as the underlying factor and named the constellation of abnormalities Syndrome X. Reaven did not include abdominal obesity, which has also been hypothesized as the underlying factor, as part of the condition.

# PREVALENCE OF METABOLIC SYNDROME

Highest Prevalence worldwide is in native Americans with nearly 60%of women ages 45-49 and 45% of men ages 45-49 meeting national cholesterol education program ,adult treatment panel III.

Based on data from the national health and nutrition examination survey the age adjusted prevalence of metabolic syndrome in united states is 34% for men and 35% for women.

Prevalence of metabolic syndrome in patients with coronary heart disease is 50% [2]

Prevalence of metabolic syndrome in patients with premature coronary artery disease is 37%(age<45 years).

Total prevalence of metabolic syndrome in asian Indians residing in India.

| Author,year city | Prevalence |
|---|---|
| Kasliwal et al;2005,Delhi | 28.5 |
| Gupta et al ;2004 Jaipur | 25 |
| Misra et al ;2004 Delhi | 12 |
| Ramachandran et al 2005;Chennai | 41 |

The prevalence of metabolic syndrome in patients with acute coronary syndrome in a study conducted in 2010 in middle east countries was around 46%.

National cholestrol education panel 2001 adult treatment panel III provided a new definition for metabolic syndrome according to which a person must have three of the following five abnormalities.

Central obesity : waist circumference >102 cm (M),88 cm (F)

Hypertriglyceridemia : triglycerides >150 mg/dl or specific medication.

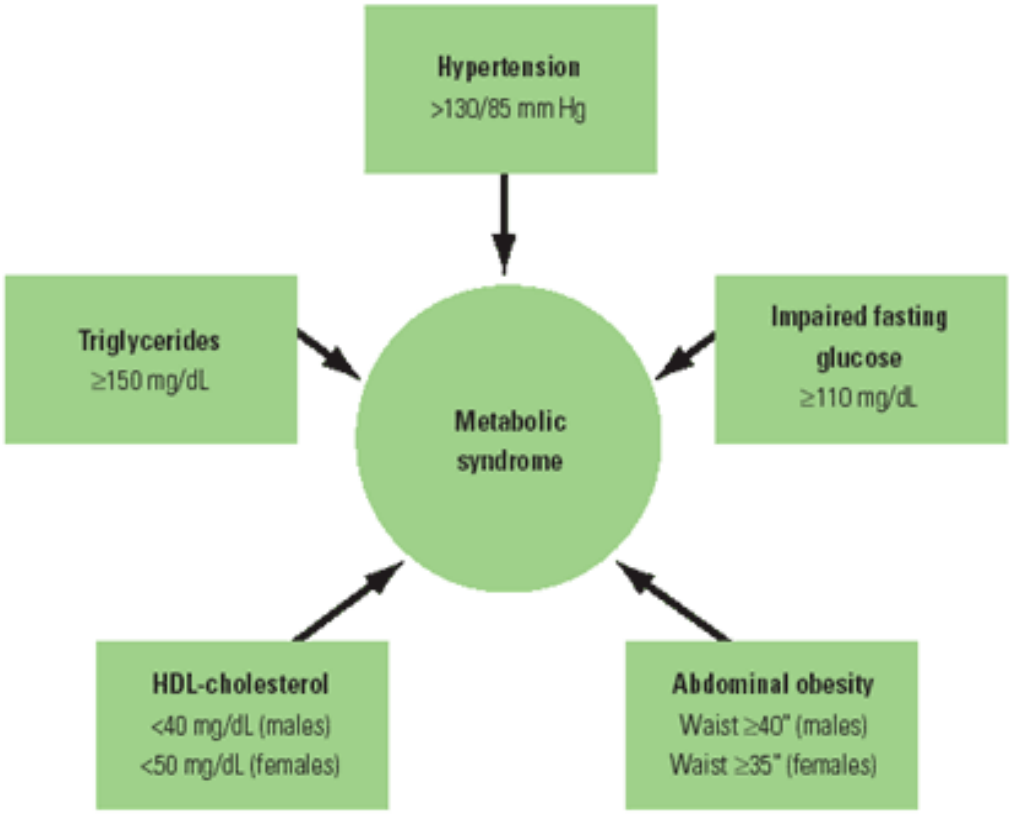
Low HDL cholesterol :<40 mg/dl and <50 mg/dl or specific medication.

Hypertension :blood pressure >130 mm systolic or.85 mm diastolic or specific medication.

Fasting plasma glucose level >100 mg/dl or specific medication or previously diagnosed type 2 diabetes.

These guidelines assert that abdominal obesity rather than elevated body mass index is highly associated with metabolic syndrome.

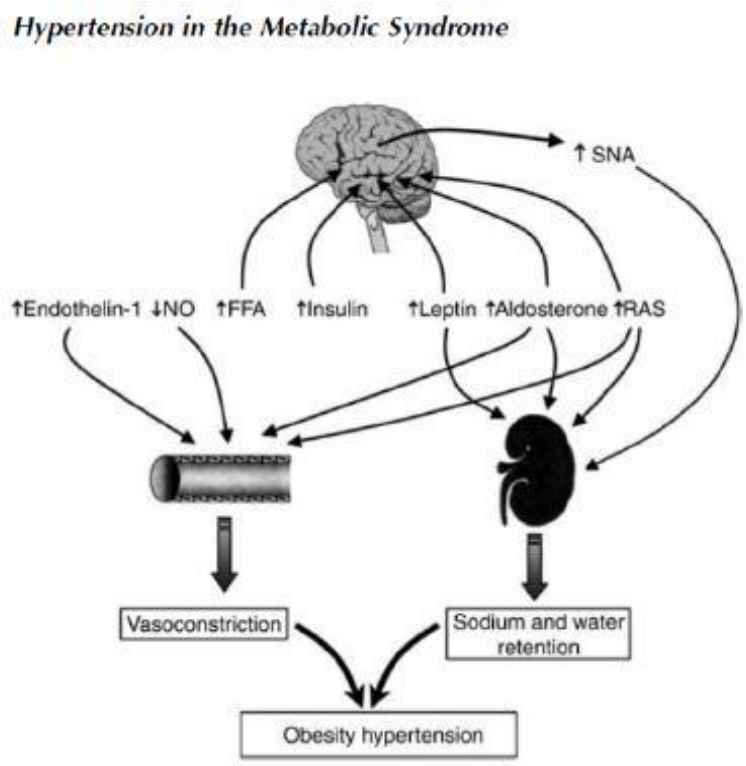
ATP III has a lower diagnostic threshold level than WHO for certain characteristics HDL cholesterol and hypertension therefore higher proportion of the population meets ATP III rather than WHO guidelines.


Hypertension
>130/85 mm Hg
Triglycerides
≥150 mg/dL
Impaired fasting glucose
≥110 mg/dL
Metabolic syndrome
HDL-cholesterol
<40 mg/dL (males)
<50 mg/dL (females)
Abdominal obesity
Waist ≥40" (males)
Waist ≥35" (females)

| IDF Criteria for Central Adiposity[a] | | |
|---|---|---|
| **Waist Circumference** | | |
| **Men** | **Women** | **Ethnicity** |
| ≥94 cm | ≥80 cm | Europid, Sub-Saharan African, Eastern & Middle Eastern |
| ≥90 cm | ≥80 cm | South Asian, Chinese, and ethnic South & Central American |
| ≥85 cm | ≥90 cm | Japanese |
| **Two or more of the following:** | | |
| Fasting triglycerides >150 mg/dL or specific medication | | |
| HDL cholesterol <40 mg/dL and <50 mg/dL for men and women, respectively, or specific medication | | |
| Blood pressure >130 systolic or >85 mm diastolic or previous diagnosis or specific medication | | |
| Fasting plasma glucose ≥100 mg/dL or previously diagnosed type 2 diabetes | | |

**Obesity in Metabolic Syndrome**

Excess adipose tissue loaded with lipids (obesity) produces abnormal amounts of NEFA & other adipokines like adiponectin, leptin, PAI-1, resistin, TNF-α , IL - 6 and other inflammmatory cytokines. The protective adiponectin is produced in subnormal amounts in obese persons. These abnormal products of adipose tissue flood various key tissues and in turn give rise to metabolic syndrome .




Cause of Hypertension in metabolic syndrome is complex and multifactorial and all of the elements of metabolic syndrome including obesity, Insulin Resistance, dyslipidemia probably are involved in mediating changes ultimately resulting in Hypertension & modifying its course

**WHO 1999** [3]

Diabetes or impaired fasting glycemia or impaired glucose tolerance or insulin resistance plus two or more of the following

Obesity-body mass index >30 kg/m2 Or

waist hip ratio>0.9 in males or>0.85 in females.

Dyslipidemia-triglycerides>150 mmol/l or HDL<0.9(males) or <1 mmol/l in females.

Hypertension-BP>140/90 mm/hg

Microalbuminuria-albumin excretion>20 micro gram/min.

| Risk Factors | IDF consensus (2005) | ATPIII criteria (2001) | WHO criteria (1999) |
|---|---|---|---|
| Obesity/abdominal obesity | Waist circumference ≥90 cm (m)*, ≥80 cm (f)** – South Asians | Waist circumference ≥102 cm (m)*, ≥88 cm (f)** | Body mass index(BMI) ≥30 kg/m2 and/or waist-to-hip ratio >0.90 (m)*, >0.85 (f)** |
| Blood pressure | ≥130/≥85 mmHg | ≥130/≥85 mmHg | ≥140/≥90 mmHg or on Medication |
| Fasting glucose | ≥5.6 mmol/L or pre-existing diabetes | ≥6.1 mmol/L or on medication for diabetes | Diabetes, impaired glucose tolerance or insulin resistance |
| Triglycerides | ≥1.7 mmol/L | ≥1.7 mmol/L | Triglycerides ≥1.7 mmol/L and/or HDL-C <0.91 mmol/L (m)*, <1.01 mmol/L (f)** |
| HDL cholesterol | <1.04 mmol/L (m)*, <1.3 mmol/L (f)** | <1.04 mmol/L (m)*, <1.3 mmol/L (f)** | --- |
| Microalbuminuria | Not used for diagnosis | Not used for diagnosis | Urinary albumin excretion rate ≥20 µg/min |
| Metabolic syndrome – definition | Abdominal obesity plus two or more risk factors | At least three risk factors | Diabetes, impaired glucose tolerance or insulin resistance plus any two or more risk factors |

Metabolic syndrome is also known as [4]

Metabolic syndrome X

Dysmetabolic syndrome

Insulin resistance syndrome

Reavens syndrome

Cardiometabolic syndrome

Beer belly syndrome

Etiology

Insulin resistance

Leptin resistance

Sympathetic overactivity

Endocannabinoid system overactivity

Reduced serotonergic responsivity.

**Endocannabinoid system overactivity**

Endo cannabinoid system signaling occurs in adipose tissue,liver,GIT and centrally within the brain.overactivation of endocannabinoid system lead to weight gain,lipogenesis,insulin resistance,dyslipidemia and impaired glucose haemostasis.

**Sympathetic overactivity**

Environmental factors activate brain centres causing frequent hypothalamic arousal via hypothalamic pituitary axis leading to elevated cortisol levels which induces the metabolic syndrome**.**

**Insulin resistance**

Insulin resistance is a precursor of type 2 diabetes mellitus. The homeostasis model assessment of insulin resistance takes into account both insulin levels and glycemia and is a more critical measure of Insulin resistance.

Insulin resistance has been considered to be a pre atherosclerotic and pre diabetic state.

**Leptin resistance**

Leptin the product of ob gene is a protein secreted mainly by adipose tissue which signals the size of energy stores to the central nervous system modulating the activity of hypothalamic centres involved in the regulation of food intake and energy balance..

other functions are immune function,,bone formation also enhances the aggregation of platelets.

Leptin attenuates the lipogenesis and oxidative actions of insulin,increases the beta oxidation of nonesterified fatty acids and decreases its esterification into triacylglycerol.

Leptin decreases lipogenesis and protects against tissue lipotoxicity.

High sustained concentration of leptin from the enlarged adipose tissue result in leptin desensitization

Leptin resistance also causes insulin resistance..Either leptin resistance or decreased leptin production by adipose tissue might potentially contribute to the development of metabolic syndrome.

## RISK FACTORS

### Obesity

Central adiposity is a key feature of this syndrome. However patients with normal weight may also be insulin resistant and have this syndrome (Ho et al;2001)[6]

### Sedentary life style

Physical inactivity is a predictor of cardiovascular disease and associated with metabolic risk factors.[7]

### Age

Greater percentage of women greater than 50 years have the syndrome than males. (Sung et al 2003) [8]

### Ethnicity

Higher prevalence of metabolic syndrome in non European groups, south Asians, black Africans, Hispanics (mckeinge PM 1992).[9]

### Genetic factors

Certain components of metabolic syndrome are strongly associated with genetic inheritance triglycerides and glucose intolerance. (poulsen et al 2001)[10]

### Birth weight

Low birth weight is associated with higher prevalence of metabolic syndrome.(Yarbrough et al1998)[11]

Diet-Insulin resistance is inversely associated with whole grain food, dietary fibres, cereal and fruit fibres. It is positively associated with glycaemic index. (Framingham offspring study, mckewn et al 2004)[12]

**Endocrine factors**

Hyperandrogenemia, polycystic ovarian syndrome, low total testosterone and SHBG levels, GH –IGF axis, glucocorticoid excess all predict the development of metabolic syndrome and diabetes (laaksonen et al).[13]

**Inflammation**

Chronic subclinical inflammation is associated with insulin resistance in metabolic syndrome.[14]

**Alcohol**

Alcohol consumption is associated with increased triglycerides, HDL and increased blood pressure. Therefore has different effects and different aspects of metabolic syndrome.[15]

**Comorbidity**

In People with diabetes mellitus, hypertension, CHD the prevalence of metabolic syndrome is higher.[16]

Among people with mental illness notably schizophrenia the prevalence of metabolic syndrome is higher. Use of ART in HIV patients is associated with high risk of metabolic syndrome.

Lipodystrophy both acquired and genetic causes predispose to insulin resistance and to metabolic syndrome.

**Pathogenesis**

Sedentary life style and high dietary calorie intake leads to decreased free fatty acids and glucose oxidation leading onto body fat accumulation and resistance to biological action of insulin.

Obesity leads to increased secretion of proinflammatory cytokines like tumour necrosis factor alpha, interleukin -6, interleukin beta from adipose tissue. 17these cytokines causes

Decreased insulin induced suppression of glucose production.

Increased fatty acid cholesterol synthesis.

Increased hepatic VLDL production.

Increased adipocyte lipolysis. (nesto 2004)

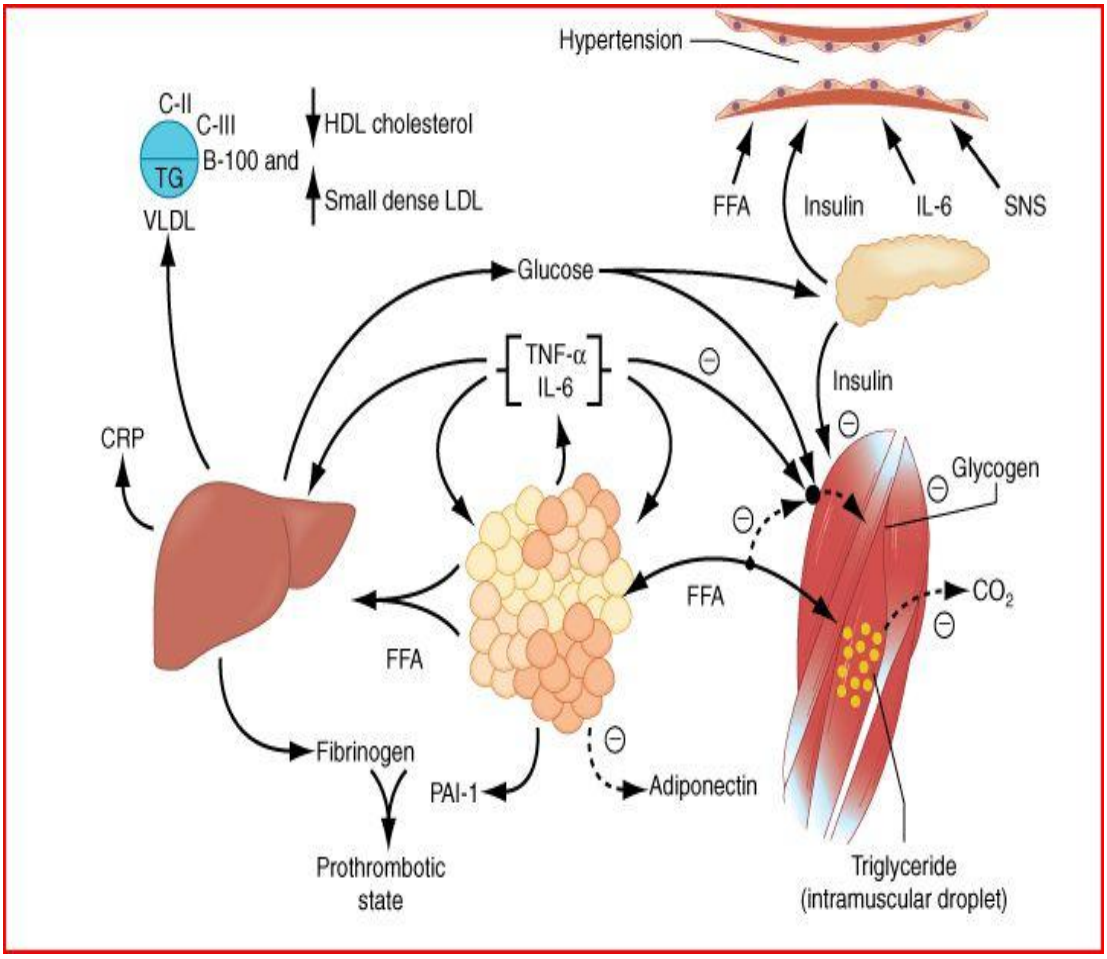
Nonesterified fatty acids released in abundance from an expanded adipose tissue mass.in the liver it tends to increase the glucose and triacylglycerol production and secretion of VLDL .

Associated abnormalities are decrease in HDL and increase in LDL cholesterol.

Nonesterified fatty acids also reduces insulin sensitivity in muscles by inhibiting insulin mediated glucose uptake leading to decreased production of glycogen, increased insulin secretion resulting in hyperinsulinemia.

Hyperinsulinemia leads to sodium reabsorption and increased sympathetic nervous system activity leading to hypertension.

Increased adipocyte lysis leads to increased non esterified fatty acids [18]


Hypertension
C-II
C-III
B-100 and
TG
VLDL
HDL cholesterol
Small dense LDL
FFA
Insulin
IL-6
SNS
Glucose
TNF-α
IL-6
Insulin
CRP
Glycogen
FFA
$CO_2$
FFA
Fibrinogen
PAI-1
Adiponectin
Prothrombotic state
Triglyceride (intramuscular droplet)

**It causes**

Increased hepatic triglyceride synthesis

Increased hepatic VLDL secretion

Reduced glucose uptake and oxidation.

Decrease in HDL

Increase in LDL

Increase in plasma glucose.

Effect of adiponectin in metabolic syndrome

Adiponectin secreted by adipocytes have its receptors in skeletal muscles and liver .It has beneficial effects on insulin sensitivity, fat and glucose metabolism.

It decreases the inflammatory pathway by reduction of nuclear factor kappa activity. (Chandran et al) [19]

Insulin resistance directly leads to endothelial dysfunction by increasing expression of ICAM 1 and thereby increasing macrophage attachment to endothelium. [20]

**Glucocorticoids**

Many of the properties of glucocorticoid hormones are antagonistic to the action of insulin. (filipovsky et al)[21]

Elevated plasma cortisol concentration are associated with high blood pressure, glucose intolerance, insulin resistance and hyperlipidemia.

**Associations of metabolic syndrome [22]**

Visceral Obesity

Type 2 diabetes

Essential hypertension

Cardiovascular disease –myocardial infarction ,peripheral vascular disease, stroke.

Carcinoma of breast, prostate, liver and colorectal cancer.

Fatty liver

Insulin resistance

Obstructive sleep apnea syndrome

Polycystic ovary syndrome

Acanthosis nigricans

Dementia

Hyperuricemia

Microalbuminuria.

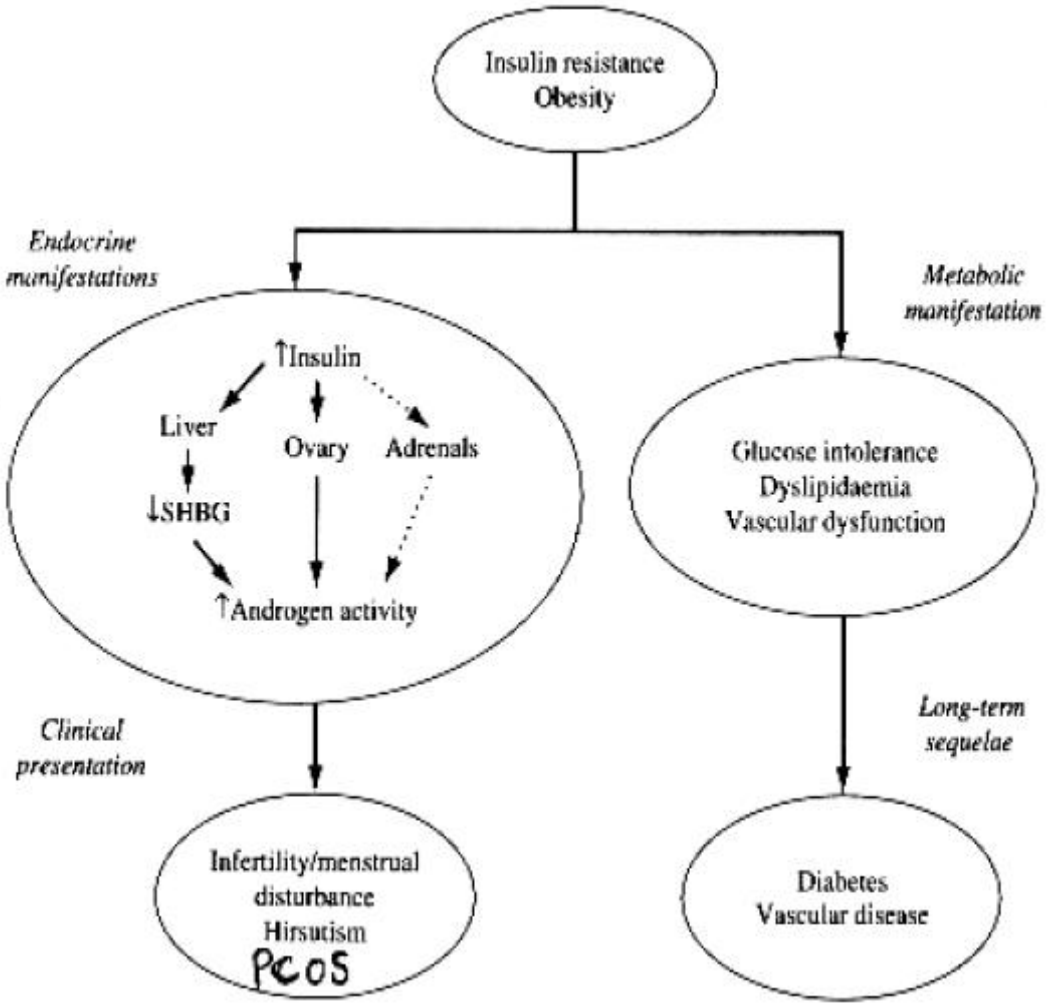
In Insulin Resistance, vasodilatory effect is lost but renal effect on sodium reabsorption is preserved. Insulin action on increasing sympathetic nervous system activity is also preserved. There is also a pathway specific inhibition of phosphatidylinositol - 3 – kinase signaling leading to imbalance in production of Nitric Oxide and Secretion of endothelin-1 contributing to decreased blood flow.

**Insulin resistance and metabolic syndrome** [23]

Inherited causes include the very rare mutation affecting the insulin receptor of post receptor signaling pathways which can lead to extreme insulin resistance. .Milder polygenic defects contribute to the Insulin resistance of type 2 diabetes . Insulin receptor mutations cause clinically distinct syndromes often with acanthosis nigricans

And In women features of polycystic ovarian disease and masculinization.

Specific syndromes include the speculatively named leprachaunism and various inherited lipodystophies in which fat is lost from subcutaneous and other depots. Recently mutations affecting the PPAR gamma gene have been shown to modify insulin sensitivity.


Insulin resistance
Obesity
Endocrine manifestations
Metabolic manifestation
↑Insulin
Liver
Ovary
Adrenals
↓SHBG
↑Androgen activity
Glucose intolerance
Dyslipidaemia
Vascular dysfunction
Clinical presentation
Long-term sequelae
Infertility/menstrual disturbance
Hirsutism
PCOS
Diabetes
Vascular disease

**Endothelial dysfunction**

In metabolic syndrome, there is abnormal Nitric Oxide metabolism that leads on to decreased endothelial Nitric Oxide production. (venugopal et al) [24] There is a reduced vasodilatory response to Insulin. (tack et al)[25]

Increased oxidative stress, inflammation and thrombosis are the consequences of vasoconstriction due to increased endothelin and Angiotensin. Abnormal nitric oxide metabolism also favours vasoconstriction.

**Inflammatory markers**

There is an increase in proinflammatory cytokines including IL-1, IL-6, IL-18, Resistin, TNFά, CRP due to overproduction by the adipose tissue derived macrophages.

Among these markers, CRP predicts the development of MetS and cardiovascular disease risk more than others.

**Obstructive sleep apnea**

Obstructive sleep apnea is commonly associated with obesity, Hypertension, Increased cytokines, Impaired glucose tolerance and Insulin resistance.

It is frequently seen in metabolic syndrome patients. continuous positive airway pressure treatment improves Insulin sensitivity.

**Atherosclerosis**

Inflammation is the bridging link between atherosclerosis & metabolic syndrome.

Chronic inflammation → endothelial dysfunction. This facilitates interaction between modified lipoproteins, monocyte derived macrophages, T cells and normal cellular elements of vessel wall, leading on to early and late atherosclerotic process.

Metabolic syndrome is a chronic low grade inflammatory condition in which inflammation impairs insulin action through various mechanisms. Other components of Metabolic syndrome also accelerates atherosclerosis.[26]

**Prothrombotic state.**

Metabolic syndrome is associated with a significant increase in the risk of developing prothrombotic state, due to disruption in the balance of factors regulating coagulation and fibrinolysis [27]

There is an increase in levels of many clotting factors Fibrinogen, Factor VII, VIII, XII & XIIIB subunit.(jugan vague et al)[28]

Fibrinolytic system is relatively inhibited due to the increase in levels of plasminogen - Activator Inhibitor I. Platelets in metabolic syndrome patients are resistant to actions of Insulin, NitricOxide and PG I2 thereby upregulating aggregation. All these, favour development of hypercoagulable prothrombotic state enhancing cardiovascular disease risk.

**Biomarkers of metabolic syndrome** [29]

| Lipid and lipoproteins | Low density lipoprotein | Vascular injury |
|---|---|---|
| Adipokines | Leptin<br>Adiponectin<br>Resistin | Modulation of insulin sensitivity .<br>Anti-inflammatory action.<br>Impairment of glucose tolerance. |
| Inflammatory markers | C-reactive protein<br>TNF alpha receptor 2<br>Interleukin -6<br>Interleukin-8 | Endothelial dysfunction<br>Insulin resistance<br>Athero thrombosis. |
| Chemokines | Monocyte chemotactic protein | Neutrophils attraction to endothelium. |
| Haemostatic markers | Plasminogen activator inhibitor 1 | Induction of cell adhesion molecule expression.<br>Insulin resistance. |

**Concept of genes predisposing to metabolic syndrome**

Mutations in the PPAR γ gene that disrupt the function of protein causes severe IR,(savage et al 2003)[30]

dyslipidemia and Hypertension

Calpain 10 gene is important in modification and processing of proteins in the cell.(sang et al)[31]

Variants in calpain gene alter the risk to Type 2 DM. Variants in SUR & kir 6.2 cause rare diabetes related disorders & predispose to Type 2 DM.[32]

Mutations in HNF1 ά. HNF-4 ά and rarely in GCK gene cause MODY.

## Genes causing metabolic syndrome

| | |
|---|---|
| Genes causing monogenic obesity | Leptin |
| | Leptin receptor |
| | Melanocortin receptor |
| | Pro-opiomelanocortin |
| Genes regulating free fatty acid metabolism | Adiponectin |
| | β-Adrenergic receptors |
| | Fatty acid binding protein-2 |
| | Lipases |
| | Uncoupling proteins |
| Genes affecting insulin sensitivity | Peroxisome proliferator-activated receptor γ |
| | Glycoprotein PC-1 |
| | Insulin receptor substrates |
| | Skeletal muscle glycogen synthase 1 |
| | Calpain-10 |
| Genes affecting lipid metabolism | CD36 |
| | Apolipoprotein E |
| | 11 β-Hydroxysteroid dehydrogenase type 1 |
| | Upstream transcription factor 1 |
| Genes related to inflammation | Tumor necrosis factor-α |
| | C-reactive protein |

Lipoproteins are complexes of lipids and proteins that are essential for the transport of cholesterol, triglyceride and fat soluble vitamins. It contains a core of hydrophobic lipids triglycerides and cholestryl esters surrounded by hydrophilic lipids phospholipids and unesterified cholesterol and proteins that interact with body fluids.[33]

Plasma lipoproteins are divided into five major classes based on their relative density.

Chylomicrons

- Very low density lipoprotein
- Intermediate density lipoprotein
- Low density lipoprotein
- High density lipoprotein.

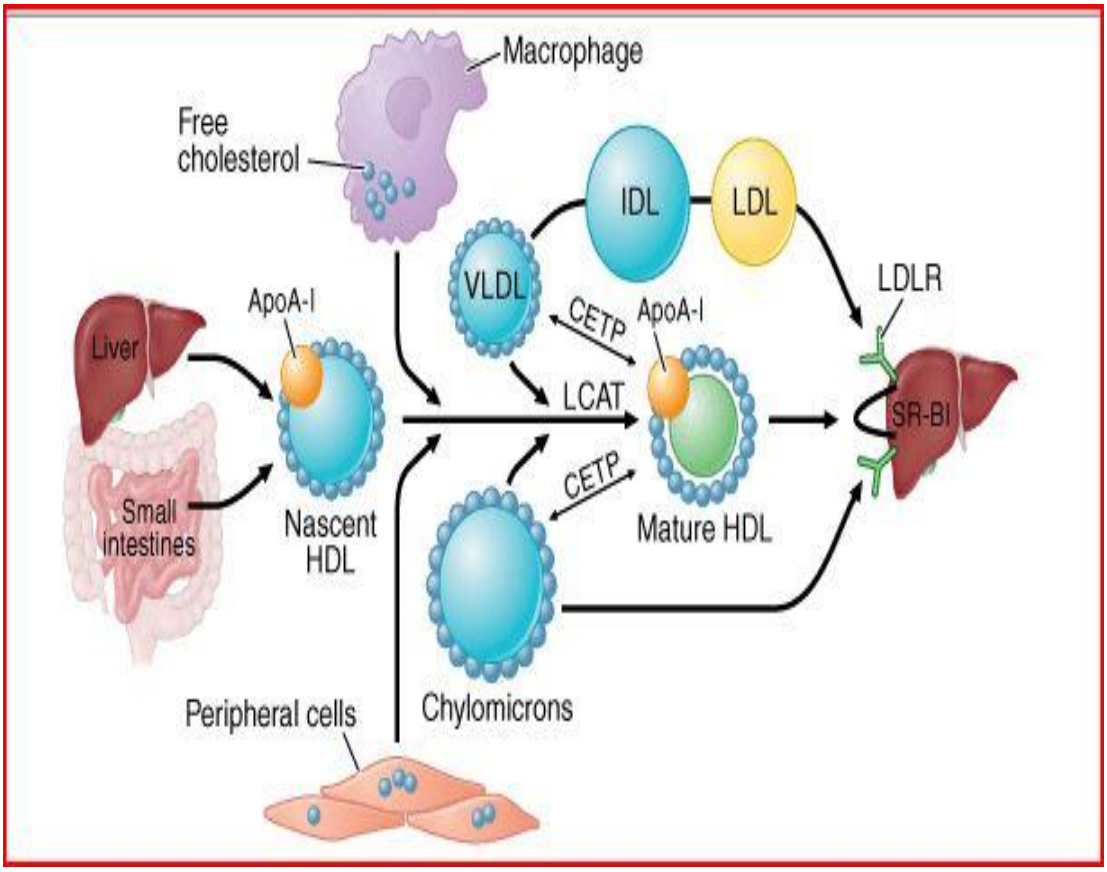
HDL is the smallest and most dense lipoprotein, chylomicron and VLDL are the largest and least dense lipoprotein.

The proteins associated with lipoproteins are called apolipoproteins they are required for the assembly structure and function of lipoproteins.

APO A1 synthesized in the liver and intestine is found in all HDL particles.

APO B 48 –contains chylomicrons

APO B 100-VLDL, IDL or LDL.


Macrophage
Free cholesterol
IDL
LDL
VLDL
LDLR
ApoA-I
CETP
ApoA-I
Liver
LCAT
SR-BI
CETP
Small intestines
Nascent HDL
Mature HDL
Chylomicrons
Peripheral cells

## Transport of dietary lipids

### Exogenous pathway [34]

Dietary triglycerides are hydrolyzed by lipases within the intestinal lumen and emulsified with bile acids.

Cholesterol and retinol are esterified to form cholestryl esters and retinyl esters.

Longer chain fatty acids are incorporated into triglycerides and packaged with APO B 48 cholestryl esters, phospholipids and cholesterol to form chylomicrons.

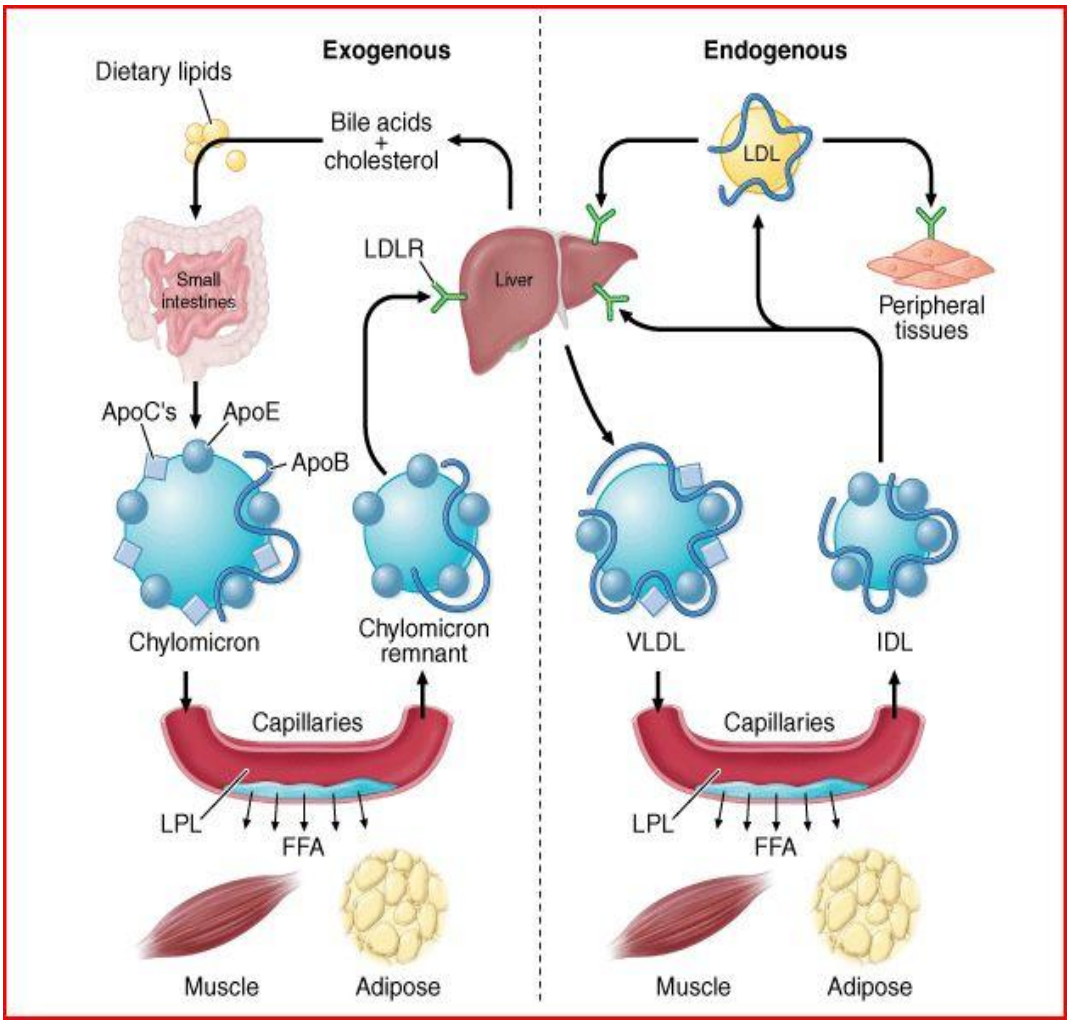
Nascent chylomicrons are secreted into intestinal lymph and delivered via thoracic duct to systemic circulation.

They become chylomicrons when nascent particles combine with apo CII and apo E derived from HDL.

The enzyme lipoprotein lipase present in capillary walls of adipose tissue cardiac and skeletal muscle hydrolyses the triacylglycerol present in chylomicrons and releases free fatty acids and glycerol.

Lipoprotein lipase is activated by CII.

The chylomicron remnants are taken up by the receptors present on the hepatocytes of the liver.


Exogenous
Endogenous
Dietary lipids
Bile acids
+
cholesterol
LDL
Small
intestines
LDLR
Liver
Peripheral
tissues
ApoC's
ApoE
ApoB
Chylomicron
Chylomicron
remnant
VLDL
IDL
Capillaries
Capillaries
LPL
FFA
LPL
FFA
Muscle
Adipose
Muscle
Adipose

**Endogenous pathway** [35]

- VLDL particles resemble chylomicrons in protein composition but contain APO B 100.
- Packaging of hepatic triglycerides with other components of nascent VLDL particle require the action of MTP.
- Triglycerides are formed due to esterification of long chain fatty acids in liver.
- After secretion into the plasma VLDL acquires APO CII and APO from HDL.
- VLDL are hydrolyzed by lipoprotein lipase especially in muscle and adipose tissue.
- They lose APO CII after which IDL is formed.
- It loses APO E and gets converted to LDL.
- LDL contains high cholesterol and less triacylglycerol.
- the cholestrol in LDL accounts for over half of the plasma cholesterol in most individuals.
- Approximately 70% of LDL cholesterol is cleared by LDL receptor mediated endocytosis in the liver.

**Investigations to be done in case of metabolic syndrome**

- Fasting lipid level
- Fasting glucose
- Liver function test.
- APO B
- High sensitivity –CRP
- Fibrinogen
- Uric acid
- Sleep study if obstructive sleep apnea is present
- Testosterone, luteinizing hormone, follicle stimulating hormone in case of polycystic ovary disease.
- Urinary microalbumin.

## Treatment of metabolic syndrome

### Life style

Weight reduction is the primary approach to this disorder. Recommendations for weight loss is calorie restriction, increased physical activity, behavioural modification.[36]

### Diet-

500 kcal restriction daily is adviced.diet restricted in carbohydrate provide a rapid initial weight loss.

### Physical activity

Increase in physical activity leads to weight reduction.60-90 min of daily activity is required to achieve this goal.[37]

### Obesity

Weight loss drugs can be used.drugs are phentermine and sibutramine.orlistat inhibits fat absorption.bariatric surgery are other options.[38]

### LDL cholesterol

- Diet restricted in saturated fats.
- HMG COA reductase inhibitors are the first choice of drugs.
- Ezetimibe is the second choice.
- Bile acid sequestrants are more effective.
- cholestyramine and cholestipol.

- Nicotinic acid
- Fenofibrates are the other drugs which can be used.

**Triglycerides**

Weight reduction

Gemfibrozil or fenofibrate is the drug of choice to lower fasting triglycerides.[39]

Other drugs are statins nicotinic acid and omega 3 fatty acid preparations.

**HDL cholesterol**

Nicotinic acid is the only available drug to raise HDL cholesterol.

**Blood pressure**

In patients with metabolic syndrome without diabetes ACE inhibitors or ARB are used.[40]

**Impaired fasting glucose**

Metformin has been showed to reduce the incidence of diabetes.

Insulin resistance Both metformin and thiazolidinediones increase insulin sensitivity.

**Blood supply of heart**

Heart is supplied by two coronary arteries arising from the ascending aorta.both arteries run in the coronary sulcus.

**Right coronary artery [41]**

Is smaller than the left coronary artery .it arises from the anterior aortic aortic sinuses.

**Branches**

Large branches

Marginal

Posterior interventricular

**Small branches**

Nodal

Right atrial

Infundibular and terminal.

**Area of distribution**

Right atrium

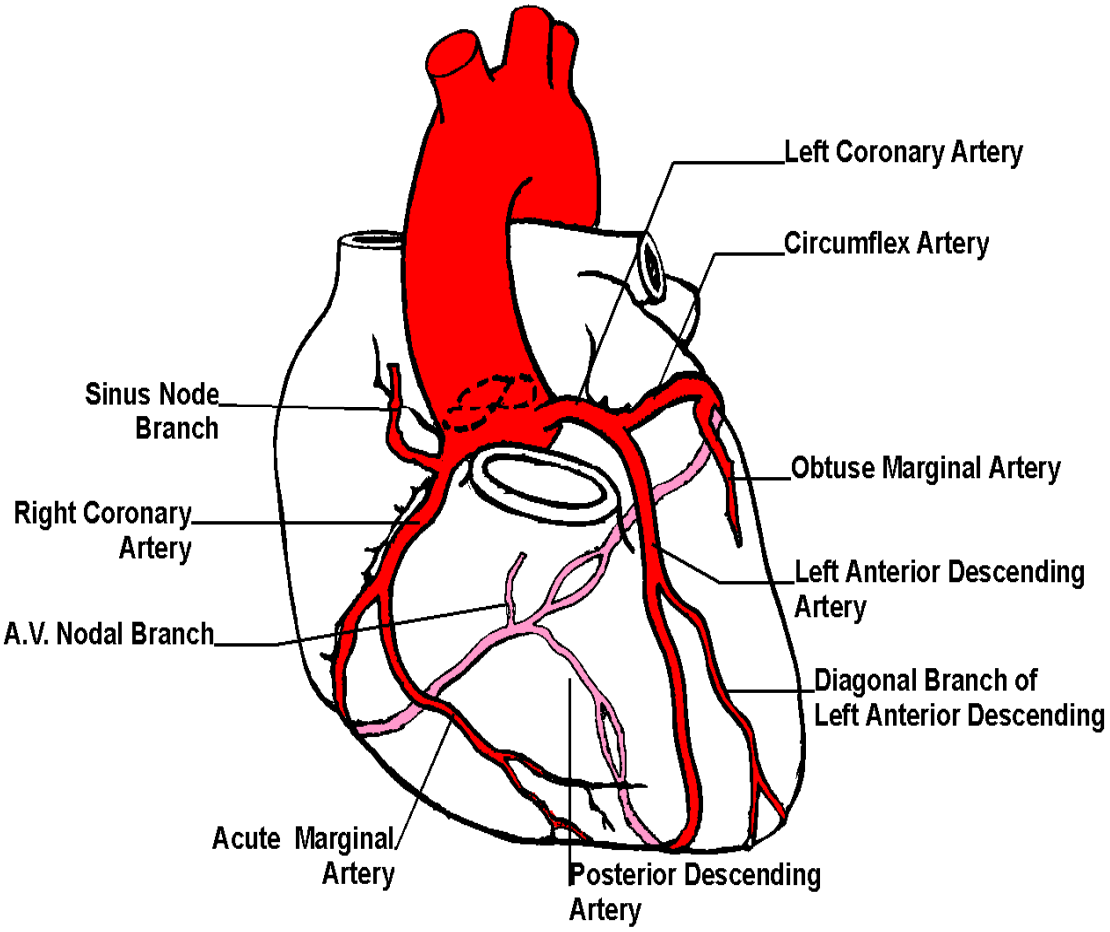
Ventricles

Greater part of the right ventricle except the area adjoining the anterior interventricular groove.

A small part of the left ventricle adjoining the posterior interventricular groove. Posterior part of interventricular septum.

Whole of the conducting system of the heart except a part of the left branch of AV bundle.

# Coronary Arteries



**Left coronary artery [42]**

Larger than the right coronary artery.arises from the left posterior aortic sinus.

**Branches**

Large branches

Anterior interventricular

Branches to the diaphragmatic surface of left ventricle.

Diagonal branch.

**Area of distribution**

Left atrium

Ventricles

Greater part of the left ventricle except the area adjoining the posterior interventricular groove.

A small part of the right ventricle adjoining the anterior interventricular groove.

Anterior part of the interventricular septum.

A part of left branch of AV bundle.

**Acute coronary syndrome** [43]

They are patients whose clinical presentation cover the following range of diagnosis.

ST segment elevation myocardial infarction

Non ST segment elevation MI

Unstable angina.

The first step is to a detailed description of the symptom complex in order to characterize the chest pain or discomfort. Five descriptors typically are considered.

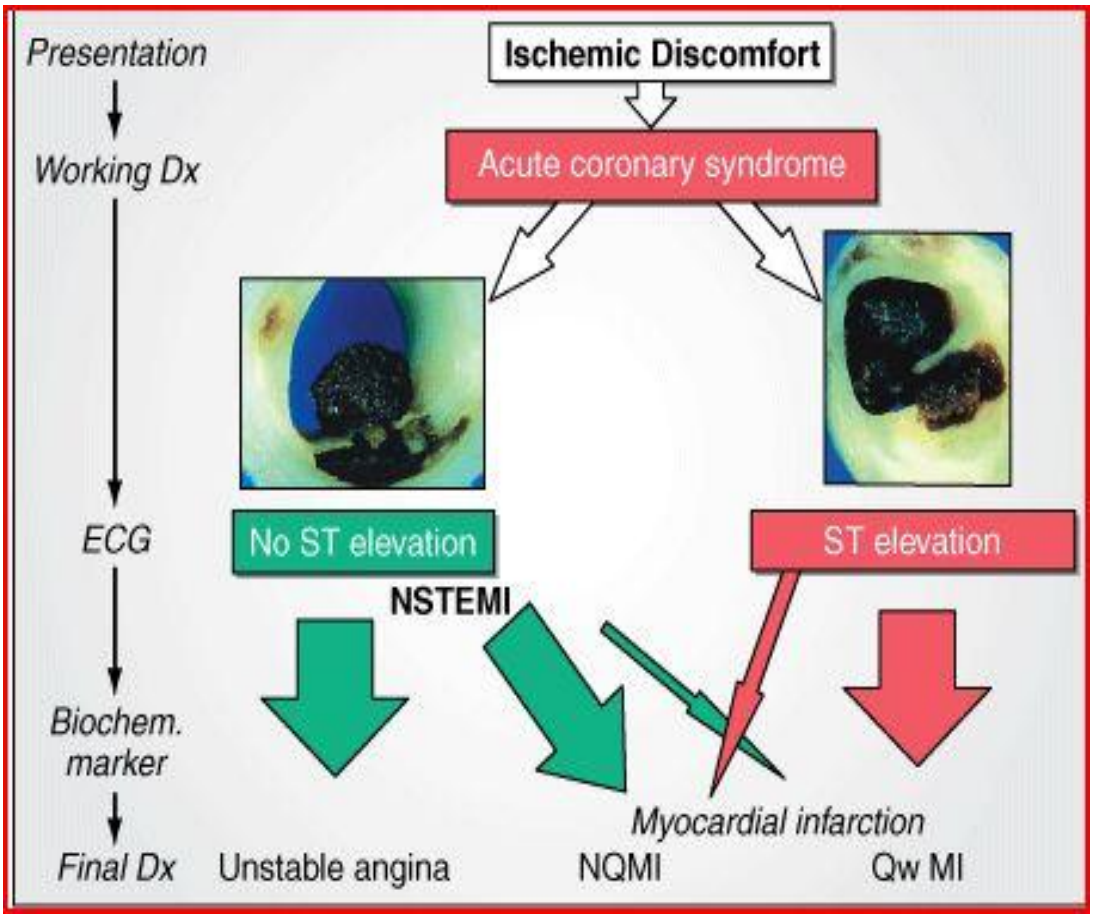
1. Location,

2. Quality,

3. Duration of the discomfort,

4. Inciting factors, and

5. Factors relieving pain


Presentation
Working Dx
ECG
Biochem. marker
Final Dx
Ischemic Discomfort
Acute coronary syndrome
No ST elevation
ST elevation
NSTEMI
Myocardial infarction
Unstable angina
NQMI
Qw MI

**Clinical classification of angina**

**Typical angina (definite)**

1. Substernal chest discomfort with a characteristic quality and duration that is
2. Provoked by exertion or emotional stress and
3. Relieved by rest or nitroglycerin.

**Atypical angina (probable)**

Meets two of the above characteristics.

Noncardiac chest pain

Meets one or none of the typical anginal characteristics.

**Stable Angina:**

Stable angina is characterized by a deep, poorly localized chest or arm discomfort (rarely described as pain) that is reproducibly associated with physical exertion or emotional stress and relieved within 5 – 15 minutes by rest or sublingual nitroglycerine, or both. The characteristics of the stable angina usually Unchanged for 60 days.

| Grade | Description 43 |
|---|---|
| Grade I | "Ordinary physical activity does not cause angina."<br>• Walking and climbing stairs.<br>• Angina with strenuous, rapid or prolonged exertion at work or recreation. |
| Grade II | "Slight limitation of ordinary activity".<br>• Walking or climbing stairs rapidly.<br>• Walking uphill.<br>• Walking or stair climbing after meals.<br>• In cold, or in wind, or under emotional stress.<br>• During the few hours after awakening.<br>• Walking > 2 blocks on the level.<br>• Climbing >1 flight of ordinary stairs at a normal pace and in normal conditions. |
| Grade III | "Marked limitation of ordinary physical activity".<br>• Walking 1 or 2 blocks on the level and<br>• Climbing 1 flight of stairs in normal conditions and at normal pace. |
| Grade IV | "Inability to carry on any physical activity without discomfort -- anginal syndrome may be present at rest." |

| Clinical Circumstances | | | |
|---|---|---|---|
| Severity | A—Develops in Presence of Extra cardiac Condition That Intensifies Myocardial Ischemia (Secondary UA) | B—Develops in Absence of Extra cardiac Condition (Primary UA) | C—Develops Within 2 wk of AMI (Post infarction UA) |
| I—New onset of severe angina or accelerated angina; no rest pain | IA | IB | IC |
| II—Angina at rest within past month but not within preceding 48 h (angina at rest, sub acute) | IIA | IIB | IIC |
| III—Angina at rest within 48 h (angina at rest, acute) | IIIA | IIIB-$T_{negetive}$<br>IIIB-$T_{positive}$ | IIIC |

UA indicates unstable angina; AMI, acute myocardial infarction.

Unstable angina is diagnosed mainly based on clinical presentation. [45]

Unstable angina is defined as angina pectoris or equivalent ischaemic discomfort with atleast one of the three features.

It occurs at rest usually lasting for more than 10 minutes.

Severe and of new onset within the prior 4-6 weeks

Occurs with a crescendo pattern.

Diagnosis of NSTEMI is established if a patient with the clinical features of unstable angina also has elevated cardiac biomarkers due to myocardial necrosis.

**Patho physiology**

Plaque rupture or erosion.

Coronary spasm

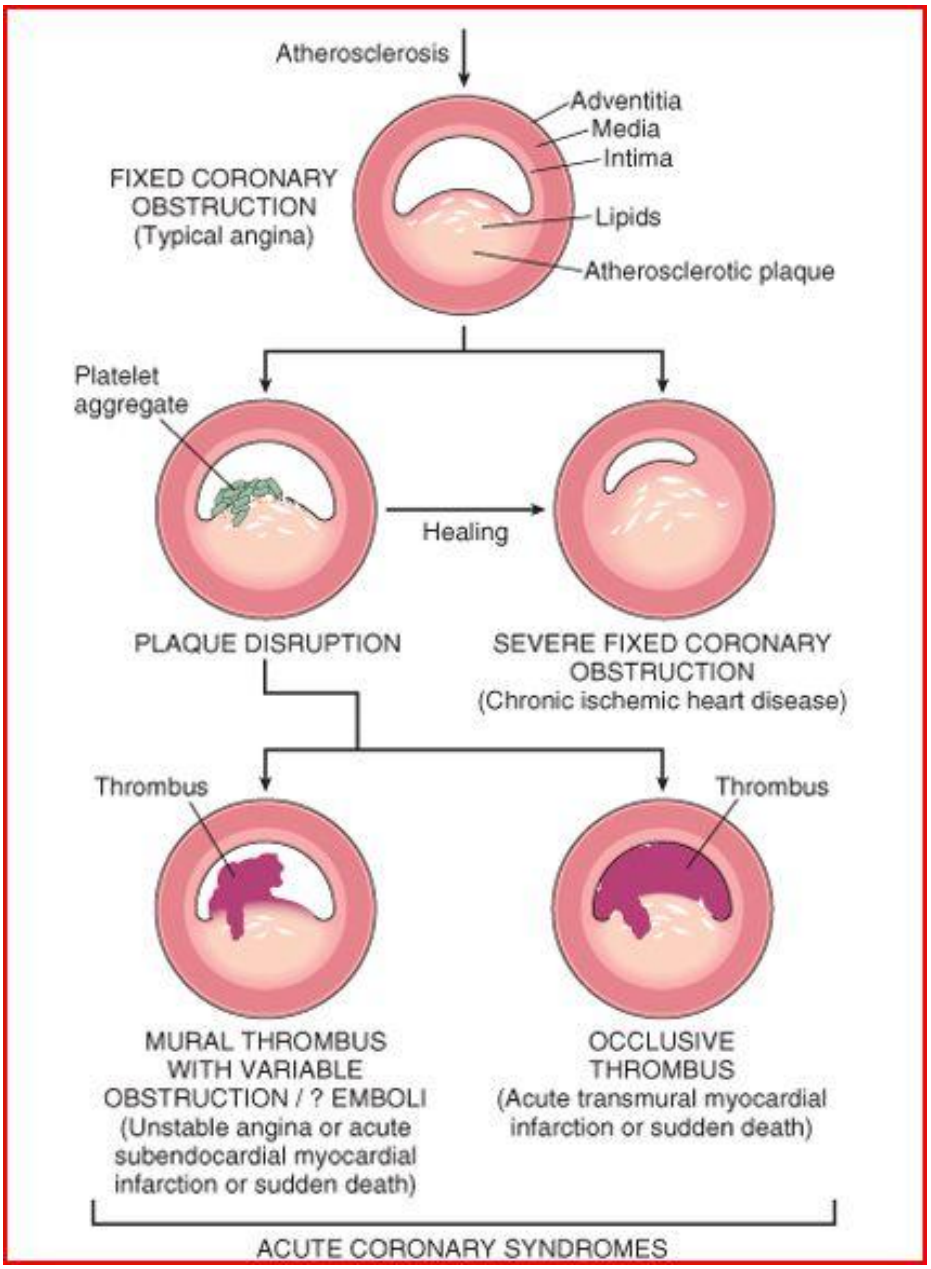
Progressive mechanical obstruction.

Secondary unstable angina.

**Clinical features**

Chest pain typically located in the substernal region or sometimes in the epigastrium that radiates to the neck, left shoulder. Anginal equivalents such as dyspnea, fatigue, epigastric discomfort, faintness and eructations..

**Crescendo Angina**: worsening of angina can be defined as symptoms that result in at least 1 Canadian Cardiovascular Society (CCS) class increase or to at least CCS Class 3 severity.


Atherosclerosis
Adventitia
Media
Intima
FIXED CORONARY OBSTRUCTION (Typical angina)
Lipids
Atherosclerotic plaque
Platelet aggregate
Healing
PLAQUE DISRUPTION
SEVERE FIXED CORONARY OBSTRUCTION (Chronic ischemic heart disease)
Thrombus
Thrombus
MURAL THROMBUS WITH VARIABLE OBSTRUCTION / ? EMBOLI (Unstable angina or acute subendocardial myocardial infarction or sudden death)
OCCLUSIVE THROMBUS (Acute transmural myocardial infarction or sudden death)
ACUTE CORONARY SYNDROMES

**Secondary Unstable Angina**:

This form of unstable angina is precipitated by an imbalance in myocardial oxygen supply and demand caused by condition extrinsic to the coronary arteries in patients with prior coronary stenosis and chronic stable angina.

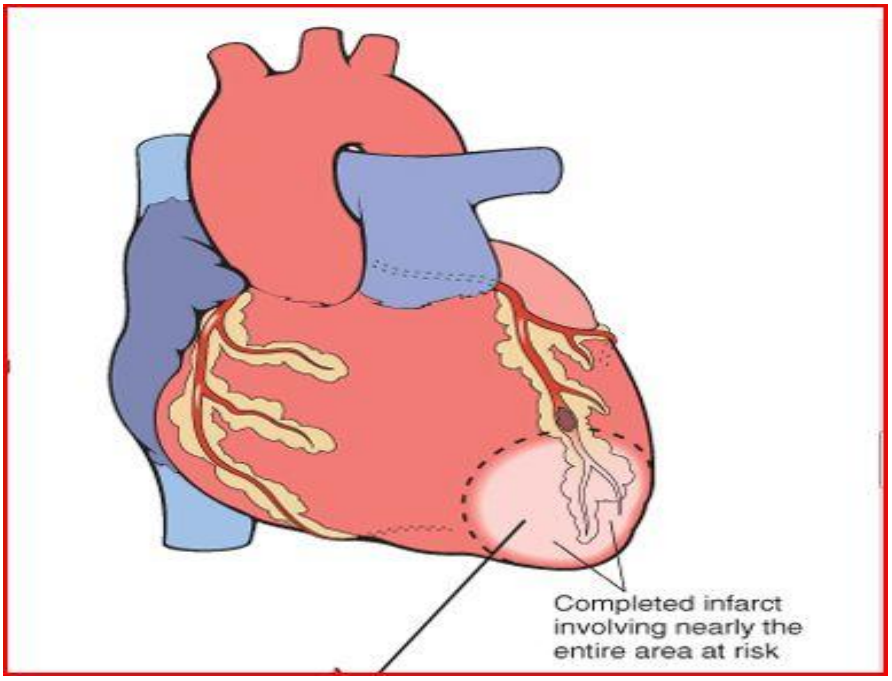
**Precipitating Factors**

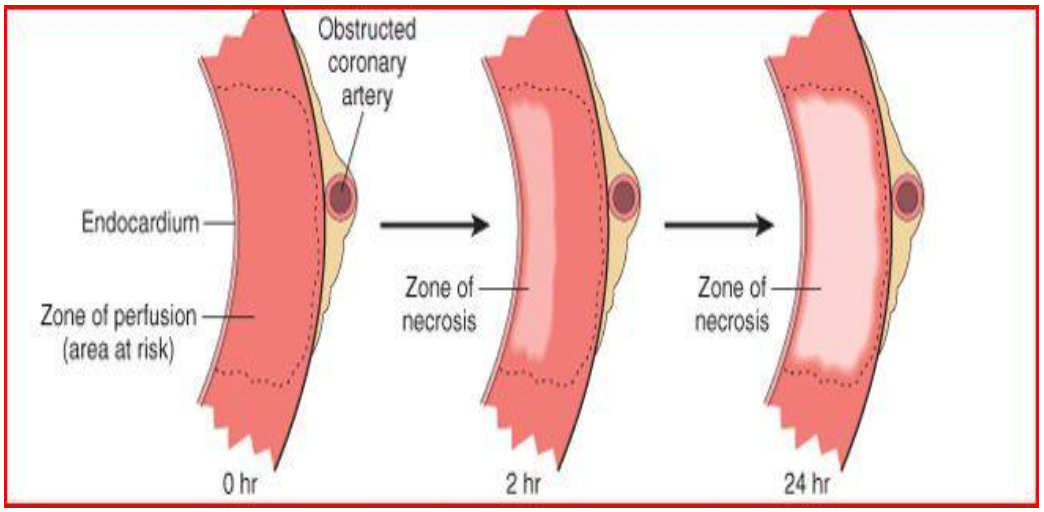
| Increased Myocardial Oxygen Demand | Decreased Oxygen Supply |
|---|---|
| • Fever<br>• Thyrotoxicosis<br>• Tachycardia<br>• Malignant Hypertension<br>• Pheochromocytoma<br>• Hypertension<br>• Aortic Stenosis<br>• High Output State<br>• Pregnancy<br>• Drugs: Cocaine, Amphetamine | • Anemia<br>• Hypotension<br>• Hypoxemia (pneumonia, CCF etc.)<br>• Carbon Monoxide Poisoning<br>• Polycythemia Vera<br>• Hyperviscosity Syndromes |

**ST elevation MI**: The classic World Health Organization criteria for an acute MI [46] require that **two of the following three elements** be present:

1. A history suggestive of coronary ischemia for a prolonged period (>30 min),
2. Evolutionary changes in serial ECGs suggestive of MI, ST segment elevation greater than 1mm in two contiguous limb leads and greater than 2mm in two contiguous chest leads.
3. A rise and fall in serum cardiac markers consistent with myonecrosis

The pain of myocardial infarction is typically substernal, diffuse, with a squeezing or pressure quality. It may radiate to the neck or jaw, shoulders, or arms. Most often, the pain is accompanied by additional symptoms, such as lightheadedness, nausea or vomiting, diaphoresis, or shortness of breath. The symptoms of myocardial infarction last longer than 30 minutes, and do not respond completely to nitroglycerin. Elderly or diabetic patients are prone to atypical symptoms, such as nausea or dyspnea as the sole symptoms of infarction. As many as one-fourth of myocardial infarctions are “silent” — that is, whatever symptoms were present did not impress the patient enough to seek medical care, or even to remember the incident.


Completed infarct
involving nearly the
entire area at risk


Obstructed
coronary
artery
Endocardium
Zone of perfusion
(area at risk)
Zone of
necrosis
Zone of
necrosis
0 hr
2 hr
24 hr

# MATERIALS AND METHODS

## METHODOLOGY:

Patients with unstable angina, STEMI and NSTEMI were evaluated for metabolic syndrome using NCEP ATPIII guidelines.

Waist circumference was measured at the narrowest point with stomach relaxed.

Blood samples were collected for fasting blood sugar with overnight fasting.

Blood samples were collected for lipid profile with 12 hours overnight fasting.

Blood pressure was recorded in right upper limb in sitting posture.

## BACKGROUND

Metabolic syndrome consist of a cluster of metabolic and haemodynamic disorders that promote the development of atherosclerosis. The presence of metabolic syndrome has been positively correlated with cardiovascular risk. The prevalence of metabolic syndrome was assessed using NCEP III.

**STUDY DESIGN:**

Prospective non randomized case series.

**STUDY POPULATION:**

About 100 patients admitted in intrinsic coronary care unit.

**INCLUSION CRITERIA:**

All patients with STEMI,NSTEMI and unstable angina.

**EXCLUSION CRITERIA:**

Patients with preexisting valvular heart disease was excluded from the study.

# RESULTS AND ANALYSIS

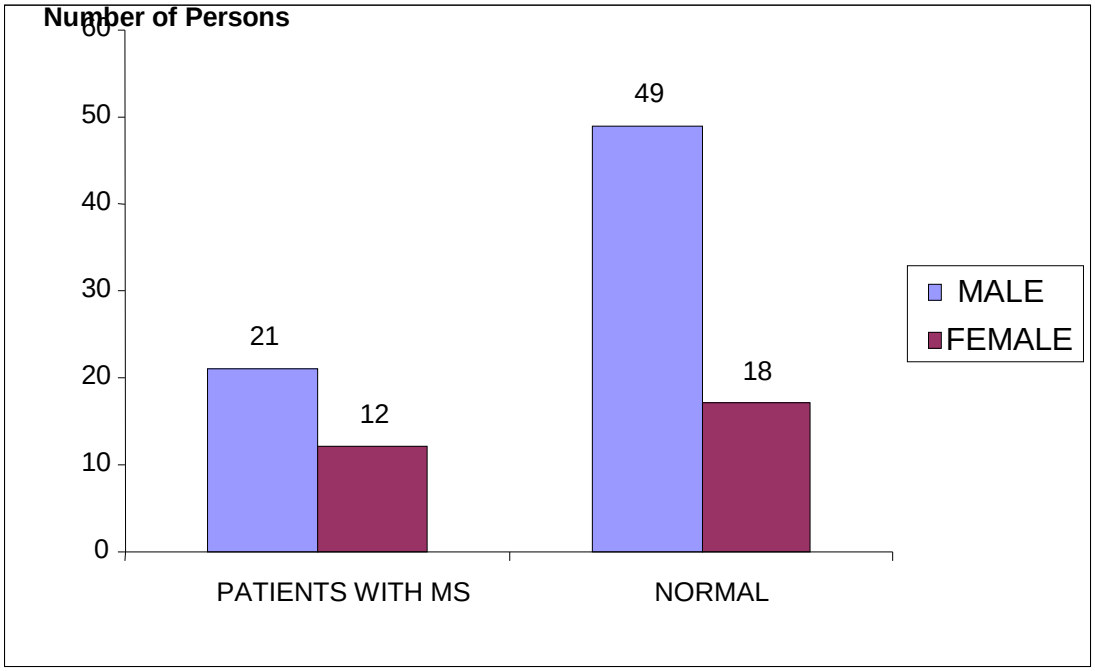
## Metabolic syndrome

| | PRESENT | ABSENT | TOTAL |
|---|---|---|---|
| MALE | 21 | 49 | 70 |
| FEMALE | 12 | 18 | 30 |

**P value not significant.**

This table shows that the study was done in 70 males and 30 females . There was a sex predilection in females when compared to males.p value was calculated using chi square test it was not significant.

## SEX PREVALENCE IN METABOLIC SYNDROME



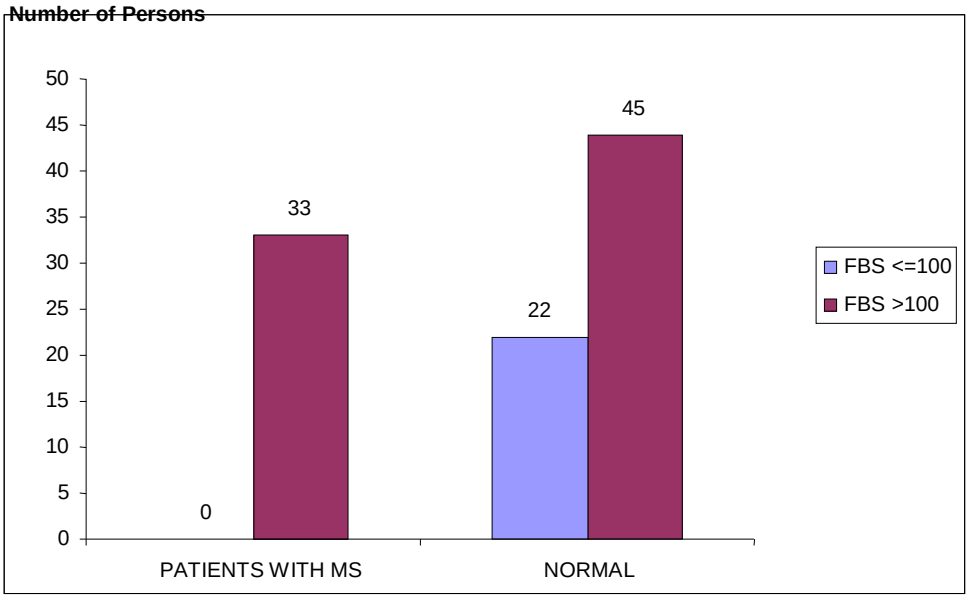
## FASTING BLOOD SUGAR AND METABOLIC SYNDROME

| | PRESENT | ABSENT | TOTAL |
|---|---|---|---|
| Fasting blood sugar <100 | 0 | 22 | 22 |
| Fasting blood sugar >100 | 33 | 45 | 78 |

P value <0.001

Fasting blood sugar values were compared in both patients with metabolic syndrome and with no metabolic syndrome .out of 33 patients with metabolic syndrome all 33 had elevated blood sugar values greater than 100 mgs%.In non metabolic syndrome group 44 patients had elevated blood sugar out of 67 patients.the percentage was 100% in patients with metabolic syndrome when compared with 65% in patients without metabolic syndrome it revealed a significant p value.

# FASTING BLOOD SUGAR AND METABOLIC SYNDROME



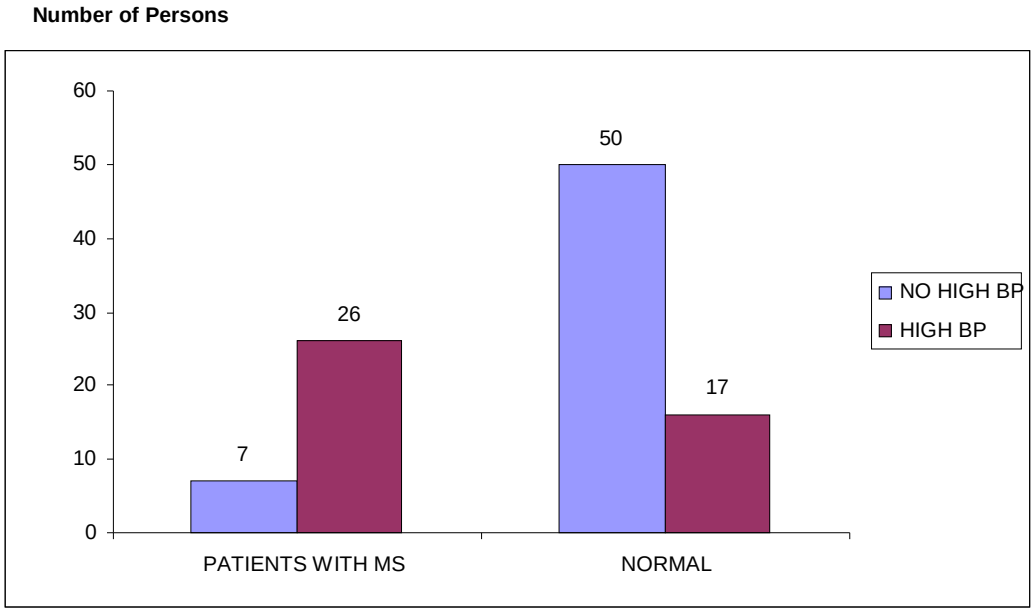
## BLOOD PRESSURE AND METABOLIC SYNDROME

| | PRESENT | ABSENT | TOTAL |
|---|---|---|---|
| BP<130/85 | 7 | 50 | 57 |
| BP>130/85 | 26 | 17 | 43 |

P value <0.001

Blood pressure was elevated more than the cut off value in 26 patients out of 33 patients with metabolic syndrome.17 patients out 67 had elevated blood pressure in patients without metabolic syndrome. The difference between the two groups were 78% in patients with metabolic syndrome and 25% in patients without metabolic syndrome. p value revealed a significant value.

# BLOOD PRESSURE AND METABOLIC SYNDROME



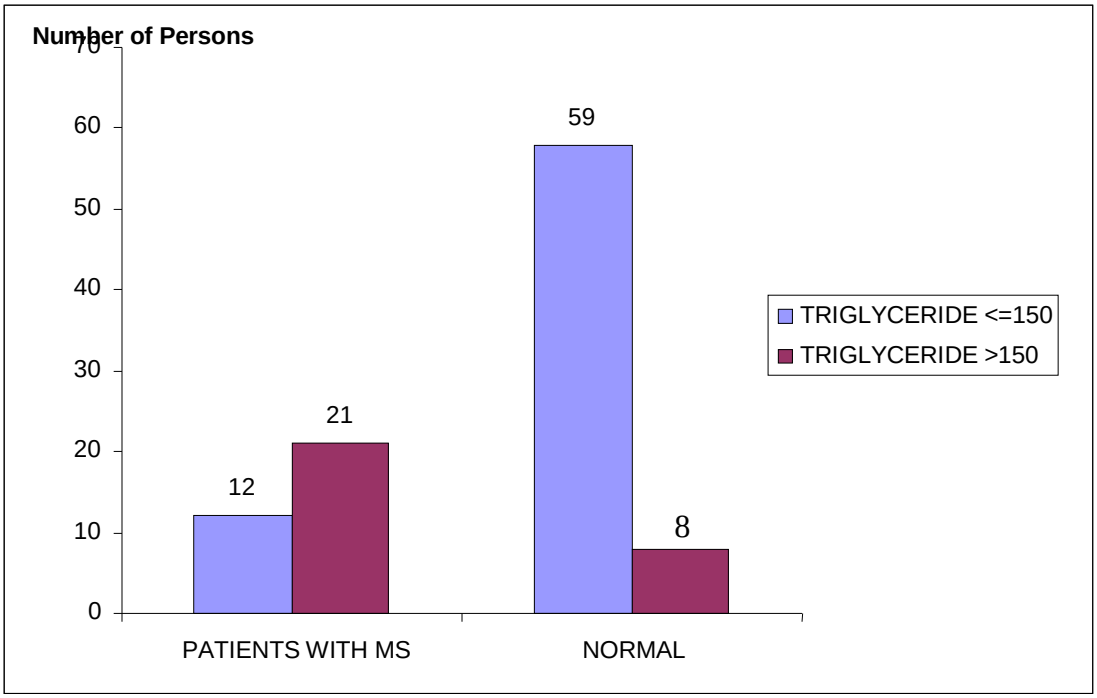
## TRIGLYCERIDES AND METABOLIC SYNDROME

| | PRESENT | ABSENT | TOTAL |
|---|---|---|---|
| TRIGLYCERIDE >150 | 21 | 8 | 29 |
| TRIGLYCERIDE <150 | 12 | 59 | 71 |

P value less than 0.001

Out of the 100 patients 21 patients had elevated triglyceride levels among 33 patients with metabolic syndrome and 8 patients with no metabolic syndrome among 67 patients. The difference between the two groups were 63%versus 13%In patients without metabolic syndrome. p value was significant.

# TRIGLYCERIDE AND METABOLIC SYNDROME



## HDL AND METABOLIC SYNDROME

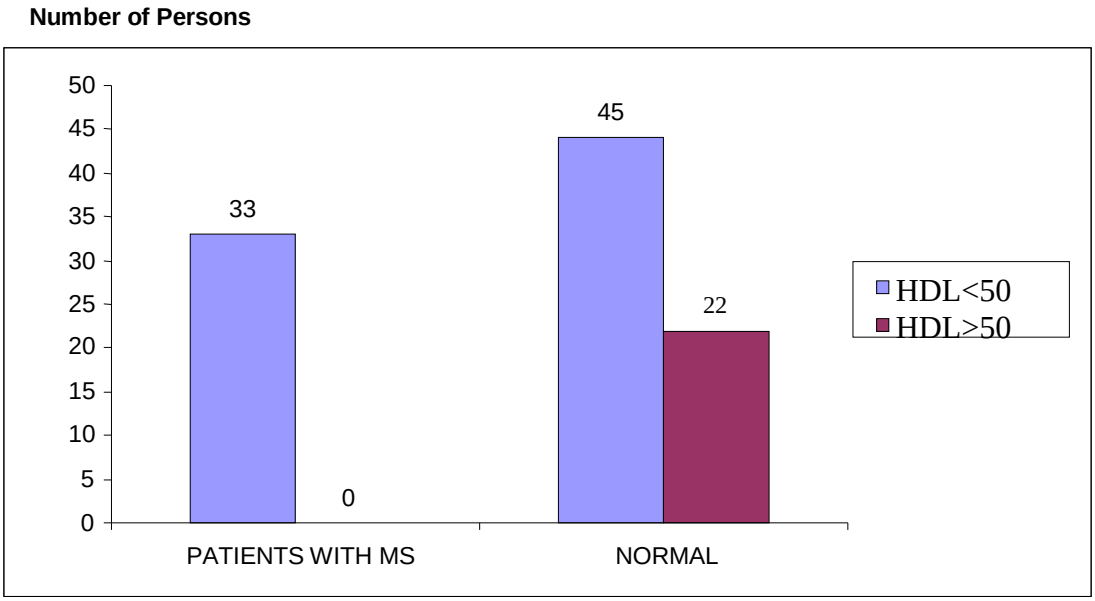
| | PRESENT | ABSENT | TOTAL |
|---|---|---|---|
| HDL >50 | 0 | 22 | 22 |
| HDL < 50 | 33 | 45 | 88 |

P value <0.001

Out of 100 patients studied with metabolic syndrome 33patients had HDL levels lesser than 50 mgs%. in patients without metabolic syndrome 45 patients had HDL levels lesser than50 mgs%.

Patients with metabolic syndrome showed100% when compared with 65% in patients without metabolic syndrome.

## HDL AND METABOLIC SYNDROME



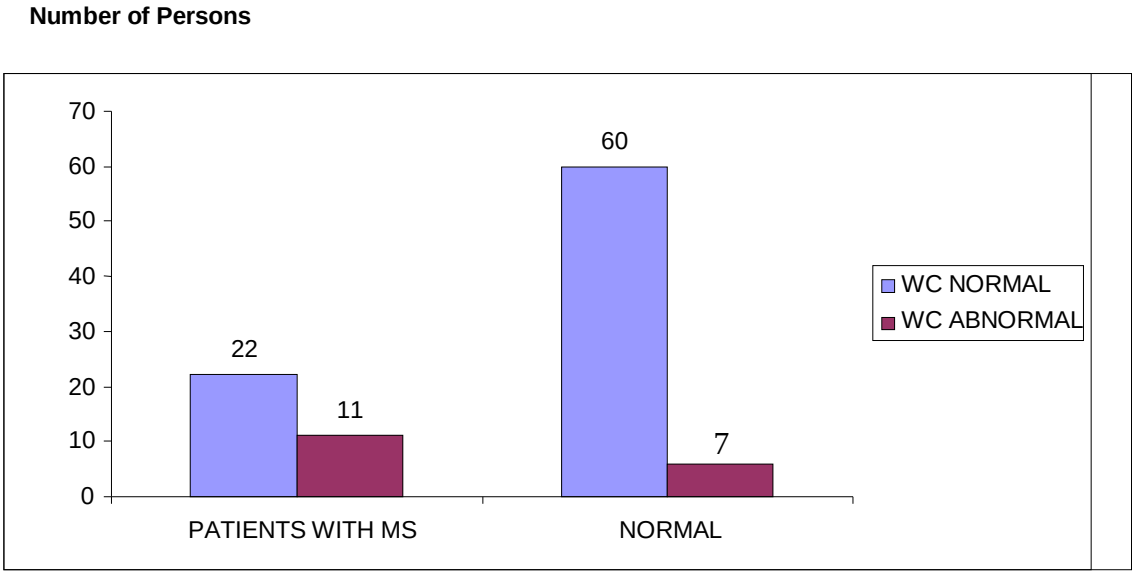
**WAIST CIRCUMFERENCE AND METABOLIC SYNDROME**

| | **PRESENT** | **ABSENT** | **TOTAL** |
|---|---|---|---|
| WAIST CIRCUMFERENCE INC | 22 | 60 | 72 |
| WAIST CIRCUMFERENCE NORMAL | 11 | 7 | 18 |

P value <0.001

Waist circumference was increased in 22 out of 33 patients with metabolic syndrome when compared to 60 out of 67 in patients without metabolic syndrome. Waist circumference in patients with metabolic syndrome was 66% when compared to 89% in patients without metabolic syndrome. p value was significant and was less than 0.001.

# WAIST CIRCUMFERENCE AND METABOLIC SYNDROME



| FACTORS | B | STANDARD ERROR | SIGNIFICANCE |
|---|---|---|---|
| FBS | -20.436 | 7569.02 | .998 |
| BP | -2.354 | 0.684 | .001 |
| TRIGLYCERIDES | -1.401 | 0.703 | .046 |
| HDL | -19.801 | 7737.78 | .998 |
| WAIST CIRCUMFERENCE | -1.511 | 0.848 | .075 |

The components highly influencing the occurrence of metabolic syndrome was studied by applying logistic regression method. .out of the 100 patients studied triglycerides and blood pressure were the two variables that were statiscally significant highly influencing the occurrence of metabolic syndrome.

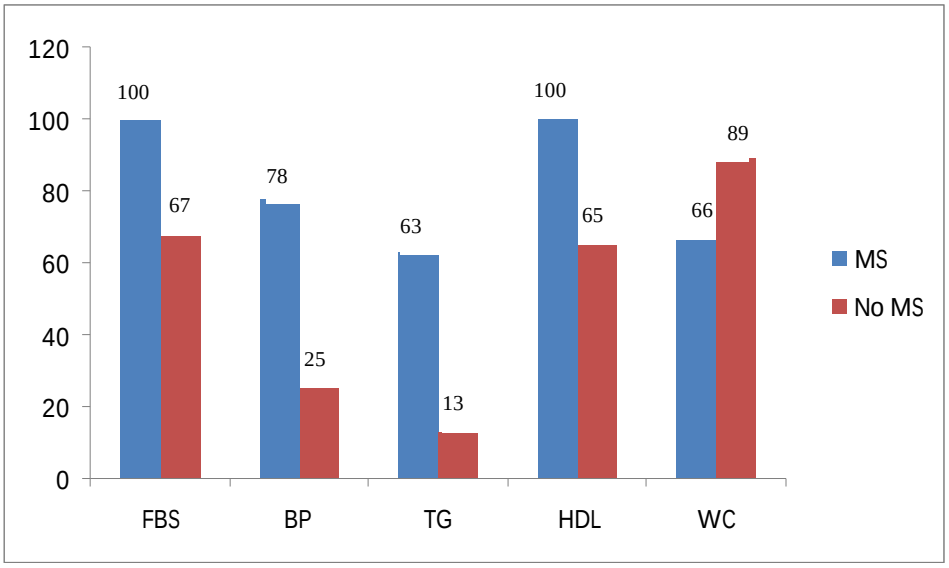
PREVALENCE OF EACH COMPONENT OF METABOLIC SYNDROME

| | **Yes** | **No** |
|---|---|---|
| Fasting blood sugar > 100 | 100% | 67% |
| Blood pressure>130/85 | 78% | 25% |
| Triglycerides>150mg/dl | 63% | 13% |
| HDL<50 mg/dl | 100% | 65% |
| Waist circumference increased | 66% | 89% |

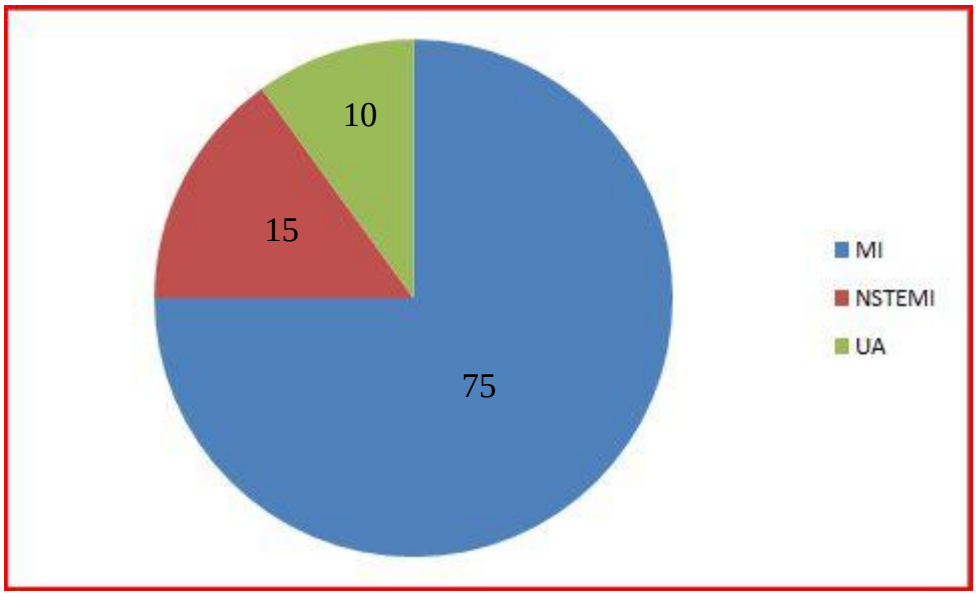
Out of the 100 patients studied in patients with metabolic syndrome all had elevated blood sugar values contributing to 100%. 26 patients had high blood pressure out of 33 contributing to 78%.21 patients had elevated triglycerides out of 33 contributing to 63%.all 33 patients had low HDL levels yielding 100%.22 patients had increased waist circumference out of 33 patients contributing 66%.

All patients showed statiscal significance confirming strong association with metabolic syndrome.

## PREVALENCE OF EACH COMPONENT WITH METABOLIC SYNDROME



# ACUTE CORONARY SYNDROME AND METABOLIC SYNDROME



## DISCUSSION

Prevalence of metabolic syndrome is highly prevalent in acute coronary syndrome patients. (Solymon BC, Bourassa MG, Campeau) [47] et al on their study on effect of increasing metabolic syndrome score on atherosclerotic risk profile and coronary artery disease.

Angiographic severity have shown an increased prevalence of metabolic syndrome 51% in their study. Similar prevalence rate was also shown by mariam zeller and coworkers in their studies.

Increased prevalence of metabolic syndrome in patients with acute coronary syndrome is also shown by two studies **one conducted in middle east countries in 2010** showed a prevalence of 46% and other **in kaunos medical university in 2008** showed a prevalence of 60%.

100 patients were studied to find out the prevalence of metabolic syndrome in patients with acute coronary syndrome .The patients were evaluated using NCEP ATP III guidelines.

75 patients with myocardial infarction, 15 patients with non STEMI, 10 patients with unstable angina were studied for the prevalence of metabolic syndrome.

The study was done in 70 females and 30 males. P value was calculated to find out the % of both sex contributing to metabolic syndrome using chi square test .p value was of no significance.there was female predilection when compared to males.

Fasting blood sugar values were compared in both patients with metabolic syndrome and with no metabolic syndrome.. out of 33

patients with metabolic syndrome all 33 had elevated blood sugar values greater than 100 mg%. In non metabolic syndrome group 44 patients had elevated blood sugar out of 67 patients. the percentage was 100% in patients with metabolic syndrome when compared with 65in patients with absent metabolic syndrome.It revealed a significant p value. This study is supported by mariamme zeller in their study [48] out of 290 metabolic syndrome patients had increased fasting glucose in contrast to 20 out of 343 non metabolic syndrome patients.

Hypertension is particularly dangerous .This concept is supported by the Framingham heart study. Blood pressure was elevated more than the cut off value in 26 patients out of 33 patients with metabolic syndrome.17 patients out 67 had elevated blood pressure in patients without metabolic syndrome. The difference between the two groups were 78% in patients with metabolic syndrome and 25% in patients without metabolic syndrome. p value revealed a significant value.

**This study is consistent with zeller study** [48] which shows 228 out of 290 in the metabolic syndrome group and 99 0ut of 343 in non metabolic syndrome patients. This **study is also similar to studies conducted by ramachandran et al in 2003 in urban asian adults.**[49]

Out of the 100 patients 21 patients had elevated triglyceride levels among 33 patients with metabolic syndrome and 9 patients with no metabolic syndrome among 67 patients. The difference between the two groups were 63%versus 13 % in patients without metabolic syndrome. p value was significant.

In the zeller study [48] elevated triglycerides was found in 57% of metabolic syndrome patients to that of 14% in the non metabolic

syndrome group. **this study is also similar to the study conducted by gupta et al in 2003 prevalence of metabolic syndrome in urban Indian population.[50]**

Out of 100 patients studied with metabolic syndrome 33patients had HDL levels lesser than 50 mgs%. in patients without metabolic syndrome 45 patients had HDL levels lesser than 50 mgs% patientswith metabolic syndrome showed100% when compared with 65% in patients without metabolic syndrome.

Low HDL is a significant risk factor MI as shown by zeller study [48] in which 80% in metabolic syndrome group 22% in non metabolic syndrome group had low HDL levels.

Waist circumference was increased in 22 out of 33 patients with metabolic syndrome when compared to 60 out of 67 in patients without metabolic syndrome. Waist circumference in patients with metabolic syndrome was 66% when compared to 89% in patients without metabolic syndrome. p value was significant and was less than 0.001. in the zeller study [48] 290 patients of metabolic syndrome group had high waist circumference when compared to 94 out of 343 non metabolic syndrome patients.

Out of the 100 patients studied triglycerides and blood pressure were the two variables that were statistically significant highly influencing the occurrence of metabolic syndrome. This was found by applying logistic regression method. Triglycerides had the highest positive predictive value in a study conducted in middle east countries[51] in 2010 and in a study conducted in 2008 at maulana azad medical college in Delhi.

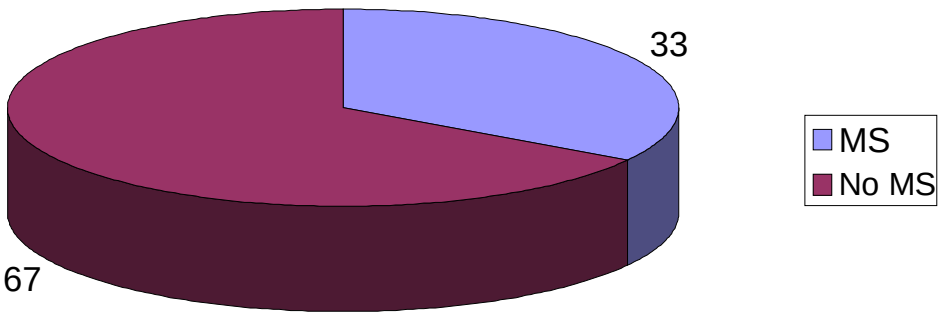
**Limitations of the study**

Our data was collected from an observational study which is a limitation.

The fundamental limitations of observational studies cannot be eliminated because of the non randomized nature and unmeasured confounding factors.

However well designed observational studies provide valid results compared to the results of randomized control trial

# TOTAL PREVALENCE OF METABOLIC SYNDROME



## Conclusion

Among 100 patients studied for the prevalence of metabolic syndrome in patients with acute coronary syndrome the overall prevalence was 33% . There was predilection in females when compared to males.

Triglycerides and blood pressure highly influenced the occurrence of metabolic syndrome.

All patients showed statistical significance confirming strong association with acute coronary syndrome.

It was also associated with increased risk of recurrent myocardial infarction. Metabolic syndrome is associated with higher risk characteristics and increased risk for the development of heart failure without increase in hospital mortality. So to prevent the complications due to metabolic syndrome there is a need for early and intensive preventive measures.

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# CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To

**Dr.A.Meenakshi, PG in MD(GM)**

Dear **Dr.A.Meenakshi, PG in MD(GM)**

The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

**"Prevalence/incidence of metabolic syndrome in patients with acute coronary syndrome as GSH "**

The following members of the ethics committee were present at the meeting held on 28.01.2008 at the Council Hall, Stanley Medical College, Chennai-1 at 10.00AM

**Dr.C.B.Tharani, Director of Pharmacology,**
**Madras Medical College, Chennai-3 - Chairman of the Ethics Committee**
**Dr.S. Chitra, Vice-Principal,**
**Stanley Medical College, Chennai – 1- Member Secretary of the Ethics Committee**

**MEMBERS**

Dr.Jayanthi
Prof.of Medical Gastroenterology
Dr.Madhavan
Prof.of Pharmacology
Dr.E.Dhandapani
Prof.of Medicine
Dr.Sujatha Sridharan
Prof.of Paediatrics
Thiru.Pachaiappan,
Junior Administrative Officer,
Thiru.A. Senthil Manoharan,
Advocate

We approve the project to be conducted in its presented form.

The Institutional Ethics Committee/Independent Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

Yours sincerely,

Chitra S

Member Secretary,
Ethics Committee

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

# PREVALENCE OF METABOLIC SYNDROME IN PATIENTS WITH ACUTE CORONARY SYNDROME AT GSH CHENNAI.

PATIENT NAME : AGE / SEX:

ADDRESS : I.P.NO.:

UNIT :

PHONE NO.:

DOA : DOD : WT:
HT:

Admission clinical diagnosis :

BMI :

RISK FACTOR : DM / SHT / CAD / CVA / CKD / PVD

: Family h/o : Y/No

: Smoker : Y/No

: Alcoholic : Y/No.

LIPID PROFILE : TC LDL HDL TG VLDL

BLOOD SUGER : RBS FBS PPBS

BLOOD UREA

CREATININE

Waist Circumference : Blood Grouping

ECG :

ECHO :

Other relevant Inv :

# ABBREVIATIONS

MS-METABOLIC SYNDROME

STEMI-ST ELEVATION MI

NSTEMI-NON ST ELEVATION MI

FBS-FASTING BLOOD SUGAR

BP-BLOOD PRESSURE

TG-TRIGLYCERIDES

WC-WAIST CIRCUMFERENCE

HDL-HIGH DENSITY LIPOPROTEIN

VLDL-VERY LOW DENSITY LIPOPROTEIN

TNF-TUMOUR NECROSIS FACTOR

PAI-PLASMINOGEN ACTIVATOR INHIBITOR

NEFA-NON ESTERIFIED FATTY ACIDS

| NAME | AGE | IP.NO | DM | HT | FBS | BP | TG | HDL | W.C | M.S |
|---|---|---|---|---|---|---|---|---|---|---|
| THIRUNAVUKARASU | 80 | 33979 | YES | NO | 142 | 110/70 | 108 | 20 | 98 | 2 |
| PARAMANANDHAM | 55 | 33669 | NO | YES | 126 | 160/100 | 249 | 22 | 78 | 1 |
| AMBIGA | 70 | 38615 | NO | NO | 90 | 130/90 | 138 | 38 | 93 | 2 |
| ARUNAGIRI | 55 | 38660 | YES | NO | 251 | 110/70 | 112 | 25 | 104 | 1 |
| INDIRANI | 50 | 38662 | YES | YES | 190 | 200/100 | 164 | 34 | 102 | 1 |
| ARUMUGAM | 70 | 33975 | NO | YES | 113 | 180/110 | 238 | 30 | 96 | 1 |
| MADIVANAN | 40 | 38218 | YES | NO | 144 | 130/100 | 104 | 50 | 64 | 2 |
| MURUGESAN | 45 | 38456 | NO | NO | 135 | 140/90 | 258 | 26 | 83 | 1 |
| DHANSEKAR | 58 | 38449 | NO | NO | 76 | 130/100 | 130 | 24 | 74 | 2 |
| SUBRAMANIYAM | 35 | 38553 | YES | NO | 159 | 130/90 | 116 | 46 | 88 | 2 |
| JEYARAMAN | 58 | 37190 | NO | NO | 86 | 120/80 | 140 | 22 | 84 | 2 |
| THIRUNAVUKARASU | 58 | 37192 | NO | NO | 121 | 130/90 | 144 | 44 | 83 | 2 |
| THIYAGARAJAN | 49 | 38058 | NO | NO | 142 | 110/70 | 164 | 34 | 90 | 2 |
| AHAMADULLAH | 58 | 38445 | YES | NO | 311 | 110/70 | 158 | 22 | 104 | 1 |
| KOTHANDAPANI | 82 | 38491 | NO | NO | 92 | 120/80 | 112 | 22 | 75 | 2 |
| GOPAL | 72 | 22686 | NO | YES | 102 | 130/90 | 127 | 58 | 97 | 2 |
| MEERA | 56 | 38530 | NO | YES | 98 | 170/100 | 110 | 62 | 88 | 2 |
| ARUMUGAM | 35 | 30395 | NO | YES | 106 | 180/110 | 133 | 40 | 70 | 2 |
| THARA | 50 | 38395 | YES | YES | 500 | 180/120 | 148 | 28 | 98 | 1 |
| CHINNASWAMY | 63 | 22687 | NO | YES | 112 | 160/90 | 132 | 32 | 92 | 1 |
| PONNUSWAMI | 65 | 38167 | YES | NO | 159 | 120/80 | 116 | 36 | 85 | 2 |
| VIJAYALAKSHMI | 54 | 22456 | NO | YES | 123 | 180/100 | 121 | 28 | 62 | 1 |
| MARY | 58 | 38185 | NO | YES | 118 | 170/90 | 128 | 30 | 90 | 1 |
| DHIVYA | 75 | 38958 | NO | NO | 103 | 130/90 | 144 | 26 | 87 | 1 |
| FRANCIS | 50 | 25836 | NO | YES | 102 | 180/90 | 177 | 32 | 87 | 1 |
| GOPAL | 60 | 25177 | NO | NO | 110 | 130/80 | 98 | 48 | 72 | 2 |
| TAMIZH | 55 | 18585 | NO | NO | 142 | 110/70 | 99 | 27 | 83 | 2 |
| MANOHAR | 60 | 19517 | NO | YES | 138 | 150/80 | 132 | 42 | 84 | 2 |
| SAMBATH | 55 | 18584 | NO | NO | 114 | 120/80 | 116 | 45 | 74 | 2 |
| RAMALINGAM | 45 | 18583 | NO | NO | 131 | 130/90 | 92 | 44 | 66 | 2 |
| PALANI | 50 | 18452 | YES | YES | 293 | 140/90 | 174 | 24 | 78 | 1 |
| ISRAEL | 60 | 36717 | NO | NO | 99 | 110/70 | 96 | 38 | 76 | 2 |
| ARULRAJ | 62 | 34013 | NO | NO | 93 | 110/70 | 90 | 36 | 70 | 2 |
| GOUSEBEE | 60 | 19517 | NO | NO | 132 | 120/80 | 112 | 24 | 84 | 2 |
| MUTHU | 54 | 18567 | NO | YES | 130 | 150/90 | 188 | 36 | 88 | 1 |
| KRISHNAVENI | 50 | 18585 | NO | NO | 142 | 140/90 | 138 | 38 | 94 | 1 |
| SENTHIL KUMAR | 65 | 37567 | YES | NO | 233 | 110/70 | 154 | 25 | 102 | 1 |
| PARUSURAMAN | 52 | 38798 | YES | YES | 184 | 180/100 | 174 | 34 | 98 | 1 |
| NARAYANAN | 45 | 37798 | NO | YES | 112 | 170/100 | 212 | 40 | 96 | 1 |
| SURESH | 65 | 18595 | NO | NO | 138 | 130/100 | 104 | 48 | 70 | 2 |
| SUNAMBEE | 48 | 18594 | NO | NO | 134 | 140/90 | 254 | 28 | 88 | 1 |
| ANNAMALAI | 70 | 18345 | NO | NO | 72 | 120/80 | 128 | 32 | 78 | 2 |
| MALLIGA | 48 | 37132 | YES | NO | 154 | 130/90 | 132 | 46 | 90 | 2 |
| JEYARAMAN | 68 | 37190 | NO | NO | 84 | 120/80 | 138 | 28 | 84 | 2 |
| VENKATESAN | 60 | 37799 | NO | NO | 124 | 130/90 | 148 | 42 | 85 | 2 |
| JEYARAMAN | 65 | 37733 | NO | YES | 148 | 150/90 | 168 | 30 | 90 | 2 |
| KANNAN | 62 | 37826 | YES | NO | 315 | 110/70 | 174 | 24 | 96 | 2 |
| SURESH | 35 | 38765 | YES | YES | 170 | 150/90 | 212 | 28 | 88 | 1 |
| KANNAIAH | 65 | 33056 | YES | YES | 220 | 140/90 | 164 | 24 | 104 | 1 |
| KUMARAN | 42 | 34561 | NO | NO | 90 | 130/90 | 114 | 26 | 76 | 2 |
| RAJESH | 55 | 38642 | NO | NO | 104 | 140/90 | 129 | 56 | 100 | 2 |
| VIJAYA | 36 | 34562 | NO | YES | 90 | 160/100 | 112 | 64 | 90 | 2 |
| KUMARAVEL | 67 | 15892 | NO | YES | 106 | 170/100 | 136 | 38 | 74 | 2 |
| KRISHNAVENI | 45 | 37264 | YES | YES | 468 | 180/100 | 154 | 22 | 98 | 1 |
| MURUGAN | 50 | 31253 | NO | YES | 114 | 150/90 | 136 | 44 | 86 | 2 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| DHINAKARAN | 70 | 17573 | YES | NO | 160 | 130/90 | 116 | 34 | 86 | 1 |
| LAKSHMI | 45 | 19853 | NO | NO | 125 | 140/90 | 168 | 28 | 88 | 2 |
| MINIYANDI | 50 | 21543 | NO | YES | 120 | 160/100 | 132 | 40 | 90 | 2 |
| RAJAPUSHPAM | 67 | 15873 | NO | NO | 102 | 120/80 | 144 | 26 | 88 | 2 |
| KARPAGAM | 65 | 19783 | NO | YES | 104 | 170/100 | 168 | 32 | 90 | 1 |
| EKAMBARAM | 72 | 37985 | NO | NO | 110 | 130/80 | 96 | 48 | 74 | 2 |
| CHINNASWAMY | 54 | 17893 | NO | NO | 140 | 110/70 | 98 | 26 | 84 | 2 |
| GOVINDASWAMY | 49 | 23546 | NO | NO | 136 | 110/70 | 134 | 30 | 86 | 2 |
| AMUDHA | 53 | 38954 | NO | NO | 116 | 130/90 | 118 | 46 | 76 | 2 |
| RAJARAMAN | 54 | 19847 | NO | NO | 130 | 130/90 | 94 | 44 | 70 | 2 |
| GNANASUNDAR | 57 | 23456 | YES | NO | 280 | 140/90 | 178 | 26 | 80 | 1 |
| MAHESWARI | 70 | 25678 | NO | NO | 100 | 110/70 | 98 | 36 | 78 | 2 |
| VELU | 67 | 30789 | NO | NO | 96 | 110/70 | 94 | 34 | 72 | 2 |
| PADMAVATHY | 55 | 30413 | NO | NO | 146 | 130/80 | 112 | 24 | 98 | 2 |
| AROKIASWAMY | 56 | 17654 | NO | NO | 124 | 120/80 | 228 | 26 | 80 | 2 |
| GOVINDARAJ | 49 | 24586 | NO | NO | 134 | 120/80 | 142 | 36 | 92 | 2 |
| MARIAMMAL | 62 | 17589 | YES | NO | 242 | 110/70 | 132 | 26 | 90 | 1 |
| BABU | 45 | 21679 | YES | NO | 198 | 210/100 | 172 | 28 | 102 | 1 |
| RAJA | 50 | 14567 | NO | YES | 102 | 160/100 | 218 | 28 | 98 | 1 |
| PANNERSELVAM | 63 | 26453 | NO | NO | 138 | 130/90 | 106 | 48 | 72 | 2 |
| PRAKASAM | 54 | 13456 | YES | NO | 142 | 110/70 | 260 | 28 | 84 | 2 |
| RATHINAVEL | 67 | 35678 | NO | NO | 78 | 120/80 | 124 | 26 | 76 | 2 |
| CHANDRA | 49 | 29867 | YES | NO | 160 | 140/90 | 118 | 48 | 90 | 1 |
| MOHAN | 49 | 31535 | NO | YES | 92 | 170/100 | 142 | 24 | 85 | 2 |
| SURESH | 74 | 19537 | NO | NO | 116 | 120/80 | 138 | 42 | 83 | 2 |
| MADHAVAN | 54 | 18756 | NO | NO | 94 | 110/70 | 114 | 24 | 76 | 2 |
| DHARMARAJ | 69 | 37564 | NO | NO | 104 | 130/90 | 125 | 59 | 95 | 2 |
| RAMESH | 70 | 14325 | NO | YES | 98 | 160/100 | 114 | 58 | 88 | 2 |
| SUBRAMANIYAM | 65 | 26745 | YES | YES | 424 | 170/100 | 145 | 28 | 97 | 1 |
| KUMAR | 50 | 34567 | NO | NO | 93 | 140/90 | 135 | 37 | 95 | 2 |
| RADHAKRISHNAN | 53 | 30567 | YES | YES | 162 | 150/90 | 117 | 39 | 86 | 2 |
| SHIVAKUMAR | 56 | 40234 | NO | NO | 125 | 120/80 | 125 | 28 | 70 | 2 |
| MUTHUKUMAR | 70 | 31234 | NO | YES | 121 | 180/100 | 131 | 33 | 92 | 1 |
| JAYANTHI | 52 | 10789 | NO | NO | 95 | 130/90 | 145 | 26 | 88 | 2 |
| KRISHNAKUMAR | 47 | 36785 | NO | YES | 110 | 170/100 | 175 | 34 | 90 | 1 |
| RAVINDRAN | 57 | 26758 | NO | NO | 118 | 120/80 | 108 | 46 | 74 | 2 |
| RAJARAMAN | 45 | 21234 | NO | NO | 96 | 110/70 | 96 | 34 | 76 | 2 |
| SARASWATHI | 62 | 17854 | NO | NO | 130 | 120/80 | 114 | 26 | 98 | 2 |
| MURUGANANDAM | 46 | 30752 | NO | NO | 128 | 110/70 | 184 | 38 | 90 | 2 |
| NAGALAKSHMI | 39 | 30175 | NO | NO | 140 | 130/90 | 136 | 40 | 86 | 2 |
| VEDACHALAM | 45 | 32134 | YES | NO | 220 | 110/70 | 164 | 28 | 100 | 1 |
| JEYAVEL | 53 | 17890 | NO | YES | 90 | 170/100 | 178 | 32 | 96 | 2 |
| THULASI | 55 | 13508 | NO | NO | 92 | 140/90 | 148 | 38 | 84 | 2 |
| PARVATHI | 59 | 30475 | NO | NO | 98 | 130/90 | 134 | 54 | 88 | 2 |
| KARPAGAM | 50 | 14575 | NO | NO | 95 | 110/70 | 132 | 52 | 88 | 2 |